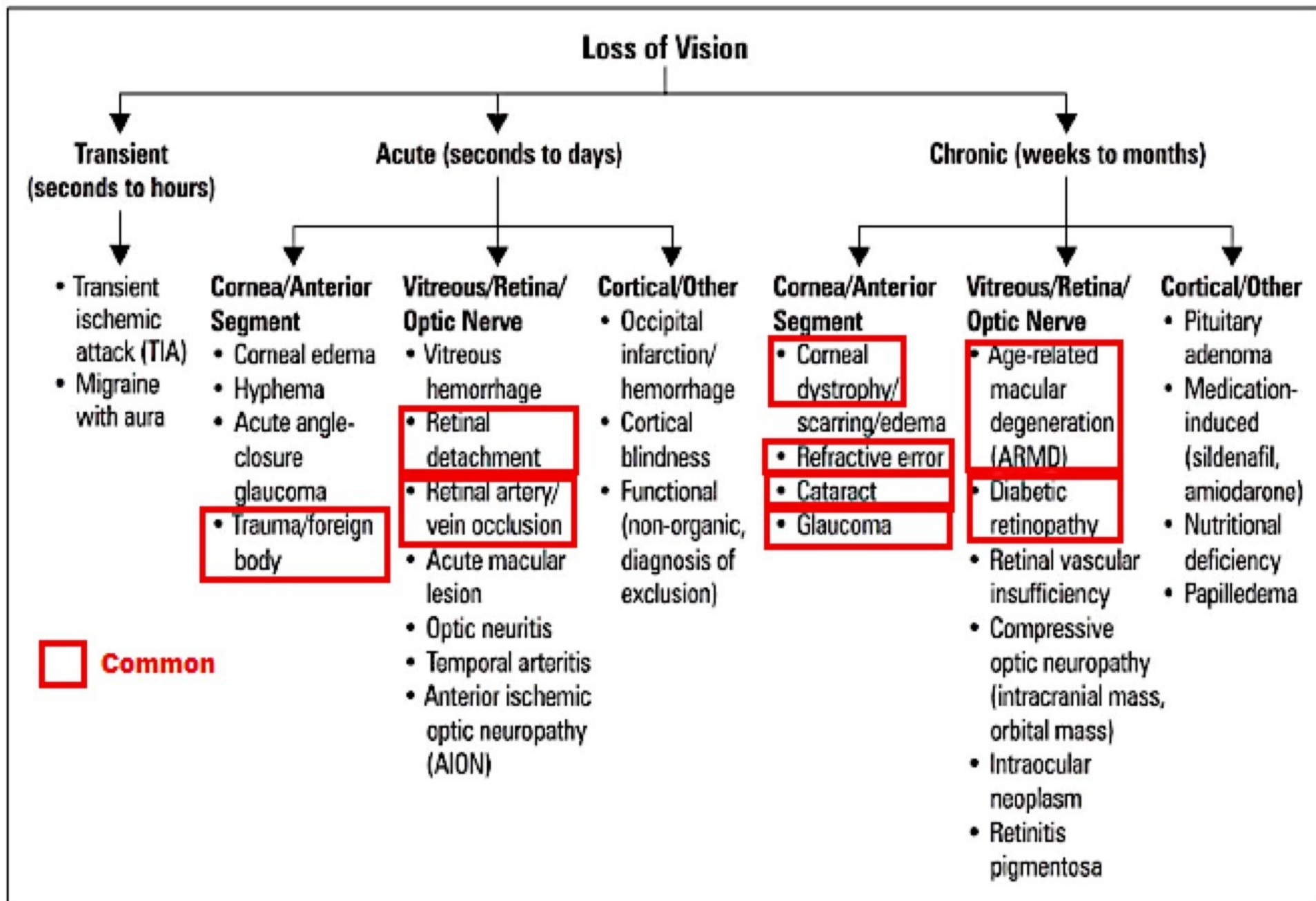




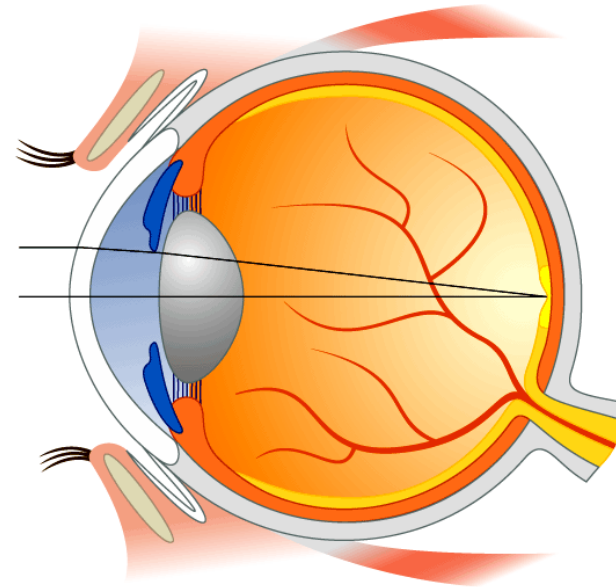
Armed Forces College Of Medicine AFCM





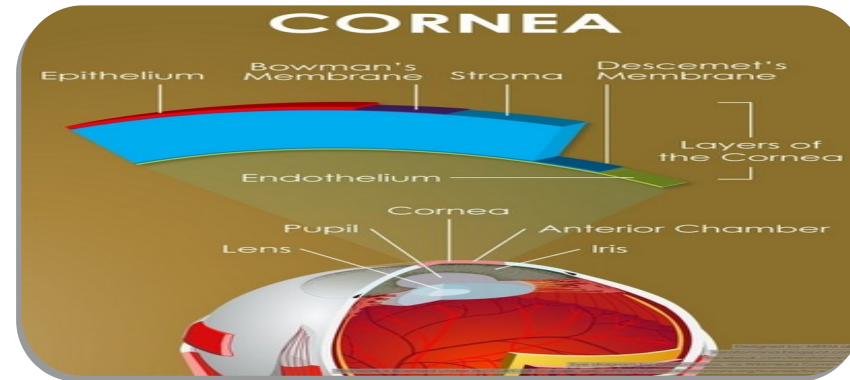
Chronic (Gradual) Loss of Vision

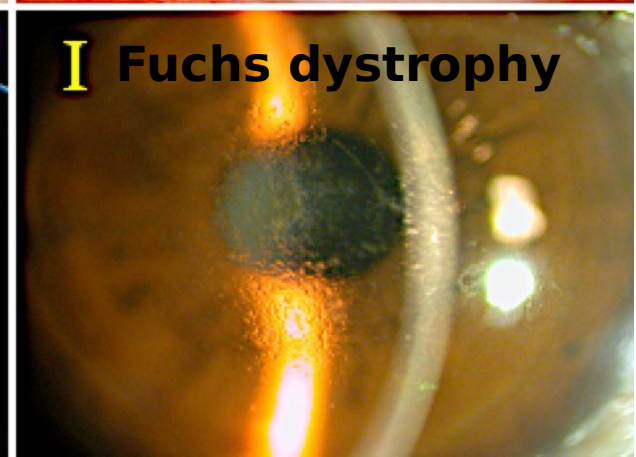
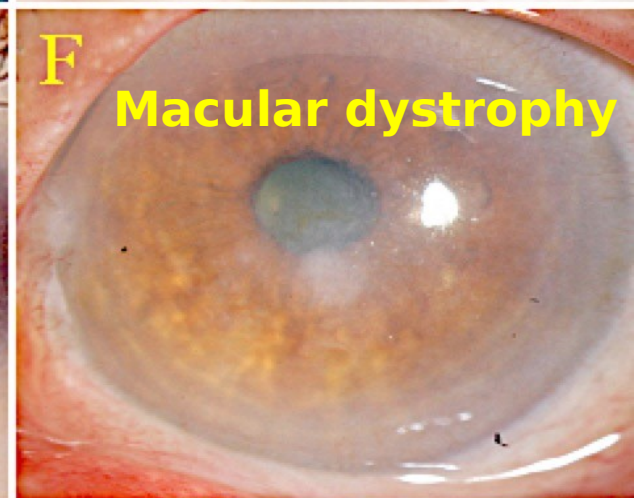
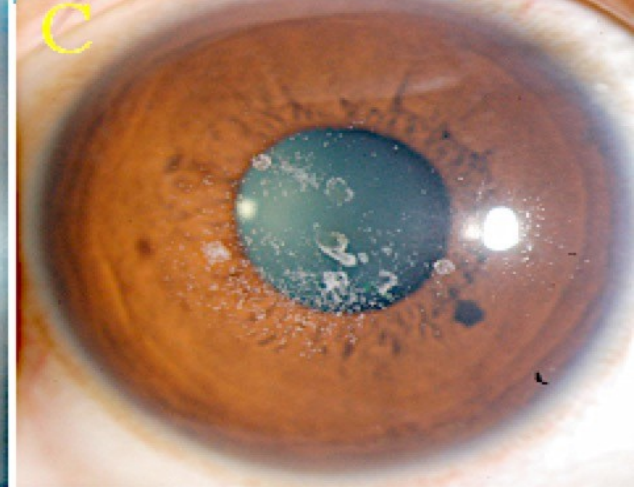
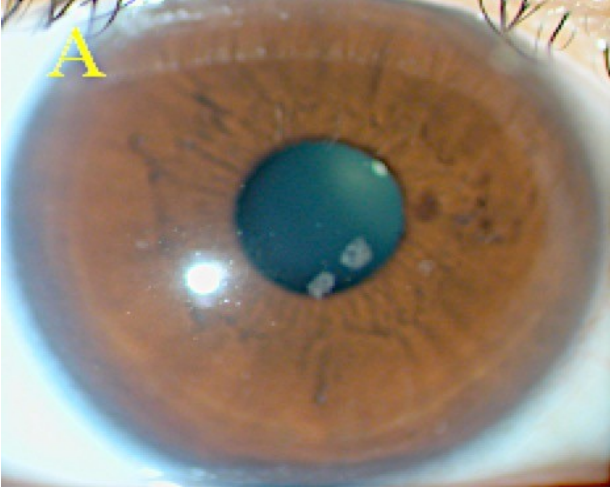
- Progressive myopia and keratoconus
- Corneal dystrophies
- Senile and **complicated** cataract
- Primary open angle glaucoma and other chronic glaucoma
- Retinal causes
 - Chronic maculopathies
 - Retinal and choroidal dystrophies
 - Dry age-related macular degeneration (ARMD)
- Optic nerve causes
 - Compressive optic neuropathy
 - Chronic papilledema
 - Hereditary optic atrophy
 - Primary optic atrophy



Corneal dystrophies

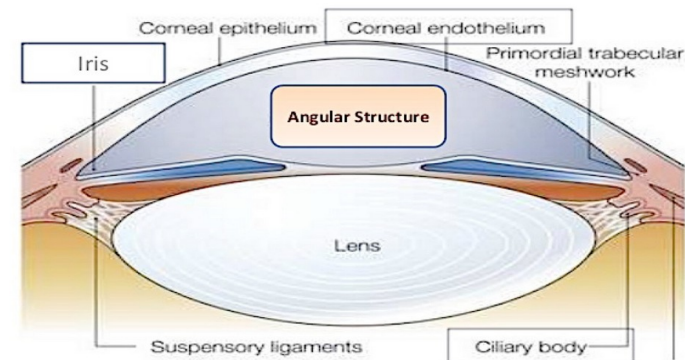
- **Bilateral** progressive corneal opacities of **hereditary** nature
- Non-inflammatory, **non-vascularized**
- Mostly local metabolic defects of proteins and MPS
- Classified into
 - Epithelial
 - Bowman's membrane
 - Stromal
 - Endothelial



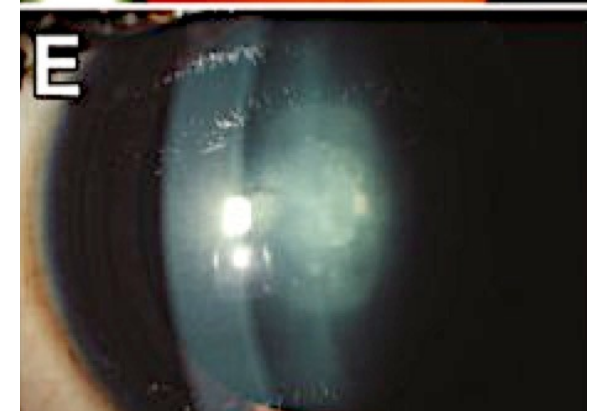
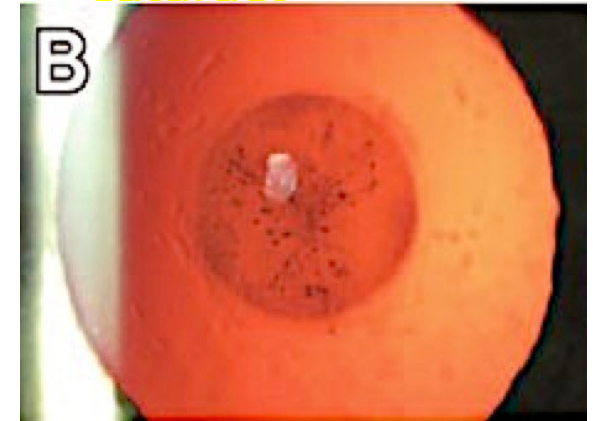
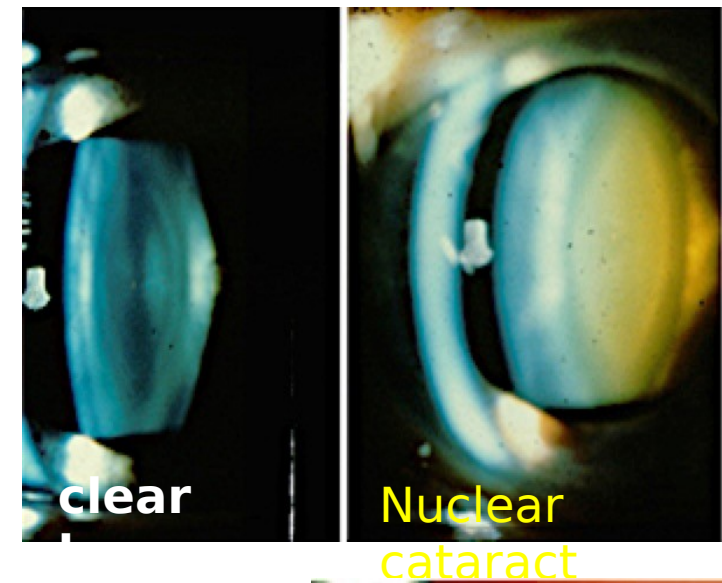


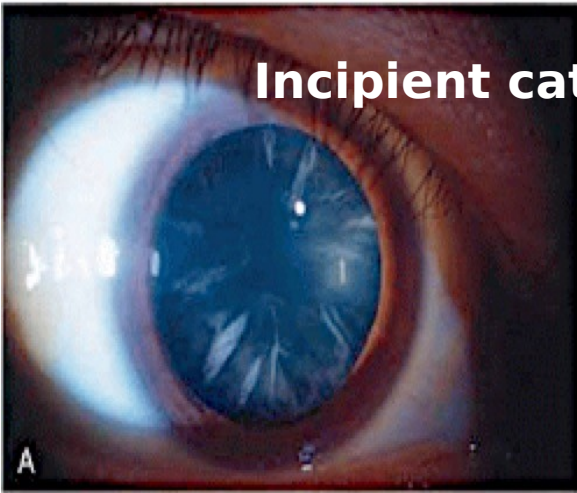
Senile cataract

- Progressive **opacification of the crystalline lens** with age due to metabolic changes from chronic UV exposure and phototoxicity بیشوف من غیر المظاره
- May be cortical or nuclear or combined.
- Cortical type has progressive **lens sclerosis** resulting in
 - **Index myopia**
 - **Nuclear hardening** (brunescant; **brown** and **black**)
 - **Dimness of light**
 - **Change of color hue**



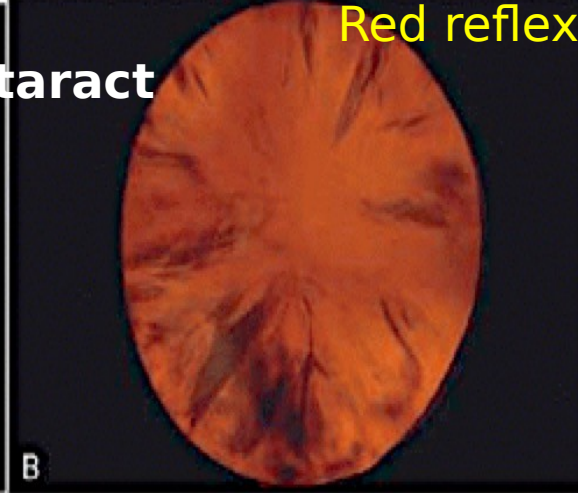
- The cortical type starts in anterior or posterior cortex and progresses from
 - Incipient
 - To immature
 - To mature
 - To hypermature
 - Some types may gallop into **intumescent** cataract
- All cataracts are surgically removed by **phakoemulsification** or **excimer laser** and an IOL is implanted
- Timing of surgery depends on visual acuity and quality of vision





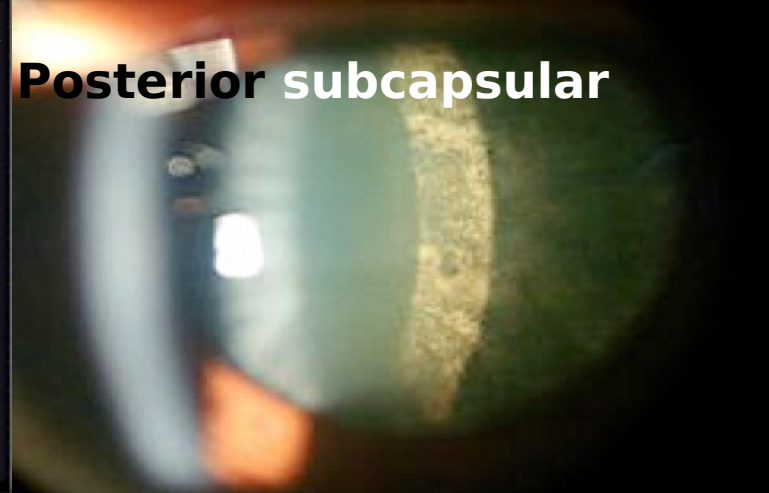
Incipient cataract

A



Red reflex

B



Posterior subcapsular



Mature cataract

EYE ROUNDS.ORG



Hypermaturation



Morganian

EYE ROUNDS.ORG

Symptoms of cataract

- Early
 - Symptoms like **errors of refraction**: indistinct far vision, difficult reading...
 - **Increased glare**
- Late
 - **Poor vision**
 - **Dimness of light**
 - **Change of color hue**
- Advanced
 - **Vision drops to HM/ good projection but never less**

Phakoemulsification

- Corneal incision (keratome knife)
- Capsulorrhhexis
- Nuclear emulsification by ultrasound or excimer laser
- Cortical aspiration
- IOL implantation



Complicated cataract

- Non-age related
 - **Local ocular disease:** chronic uveitis, chronic RD, RP, prolonged topical steroids...
 - Traumatic
 - **Systemic disease:** Diabetes, Cushing, hyper and hypoparathyroidism, severe anemia
 - Drugs: steroids, statins, heavy metals ...

Complicated cataract

- Any age
- Loss of vision may be out of proportion to the degree of cataract
- Mostly of the posterior subcapsular type with polychromatic luster
- Vision may be < HM- good projection

PRIMARY OPEN-ANGLE GLAUCOMA ^{مشن} IRIS

- Progressive Optic Neuropathy in which elevated IOP is the most important risk factor.
- If left untreated, it is a common cause of irreversible blindness , optic atrophy and characteristic **optic disc appearance** .

Primary open-angle glaucoma

- Risk factors
 - High IOP (OHT)
 - Age
 - Myopia
 - **Family history**
- **NO EARLY SYMPTOMS, NO HEADACHE, NO LOSS OF VISION** SCREENING ABOVE 40 YRS.
- IOP changes (tonometry)
 - ABOVE NORMAL RANGE (**21** mmHg)
 - Big diurnal variation
 - Difference between eyes > **2mmHg**
 - Water drinking test >6 mmHg

IOP VARIATION:

- ▣ 10 – 21 mmHg
- ▣ Is it constant through the day?

Diurnal variation:

Highest: morning

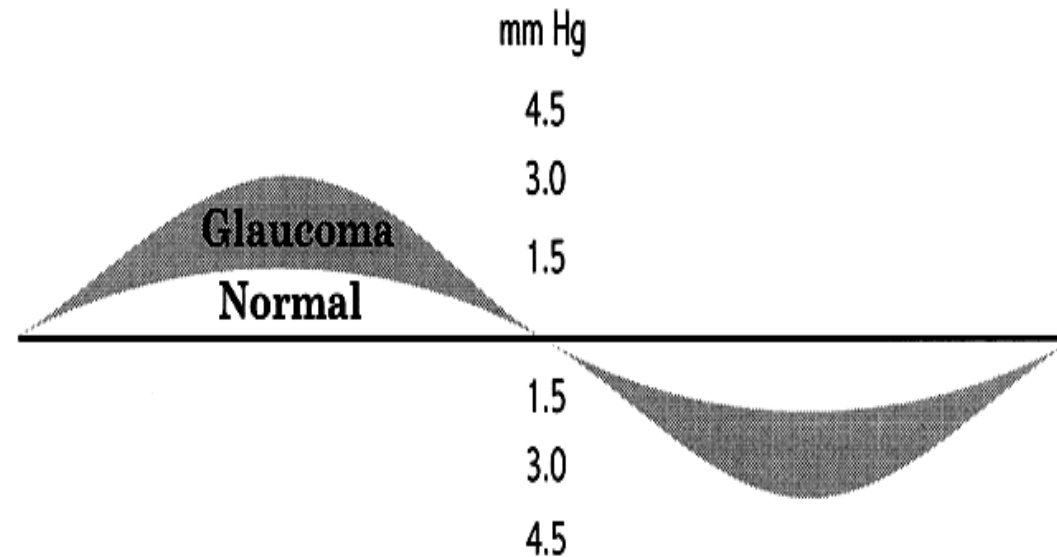
Lowest: evening

Difference max. 4 mmHg

Difference between the 2 eyes:

Not more than 2-3 mmHg

Circadian Variation in IOP



CLINICAL PICTURE:

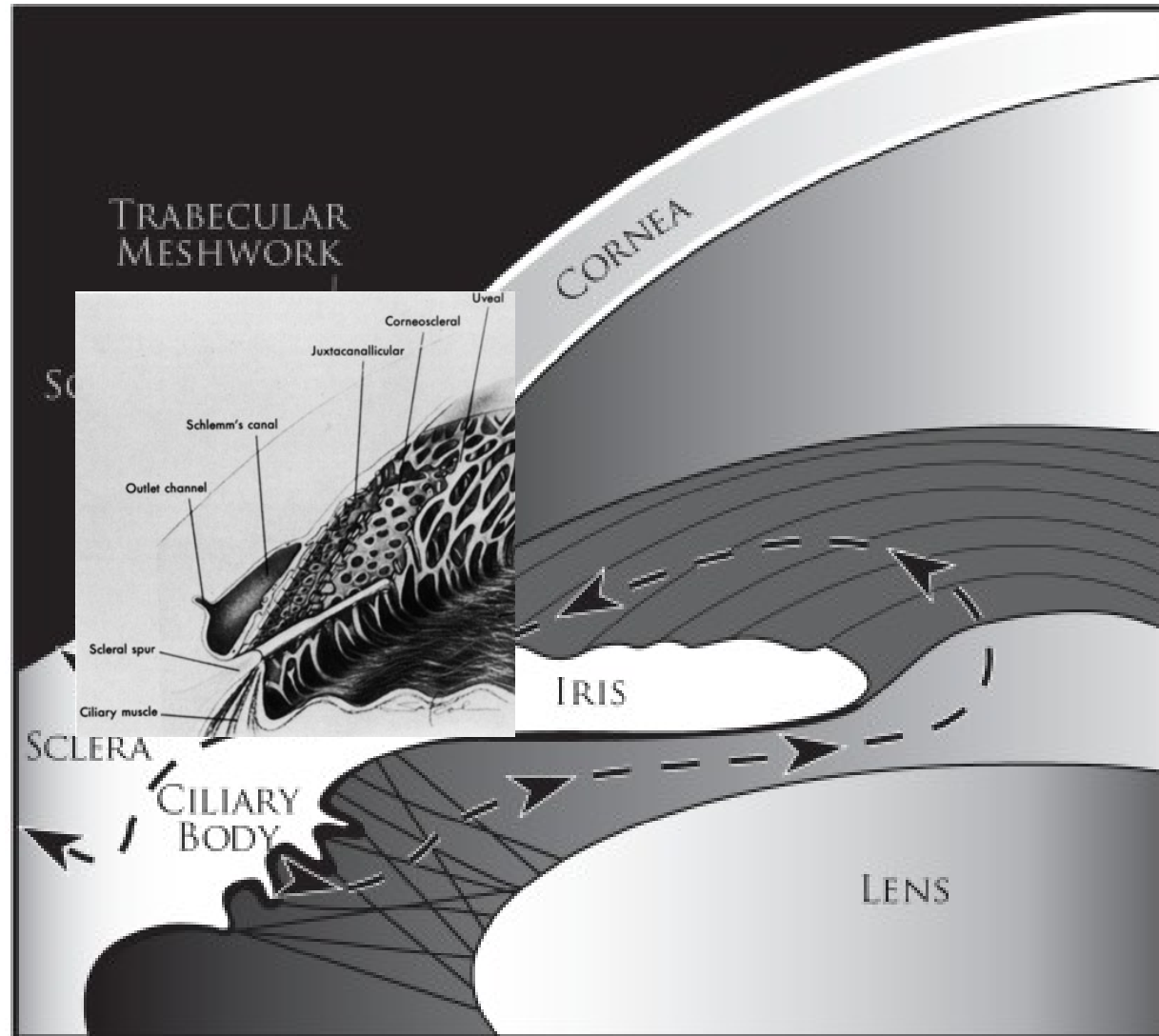
▮ Symptoms:

**Asymptom
atic**

Glaucoma is the silent thief of the vision

AQUEOUS CIRCULATION:

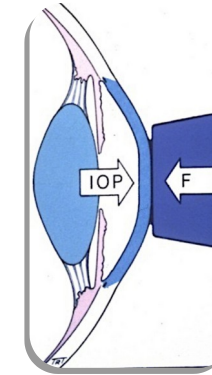
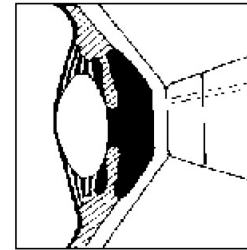
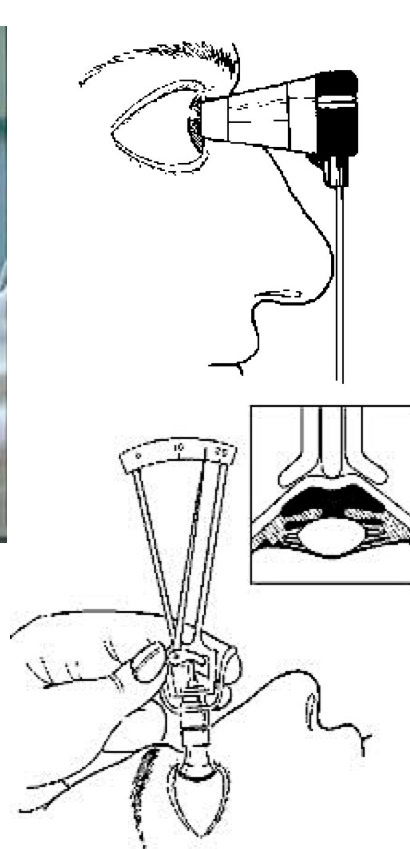
- Posterior chamber
- Pupil
- Anterior chamber
- Angle



IOP Measurements



Digital Method

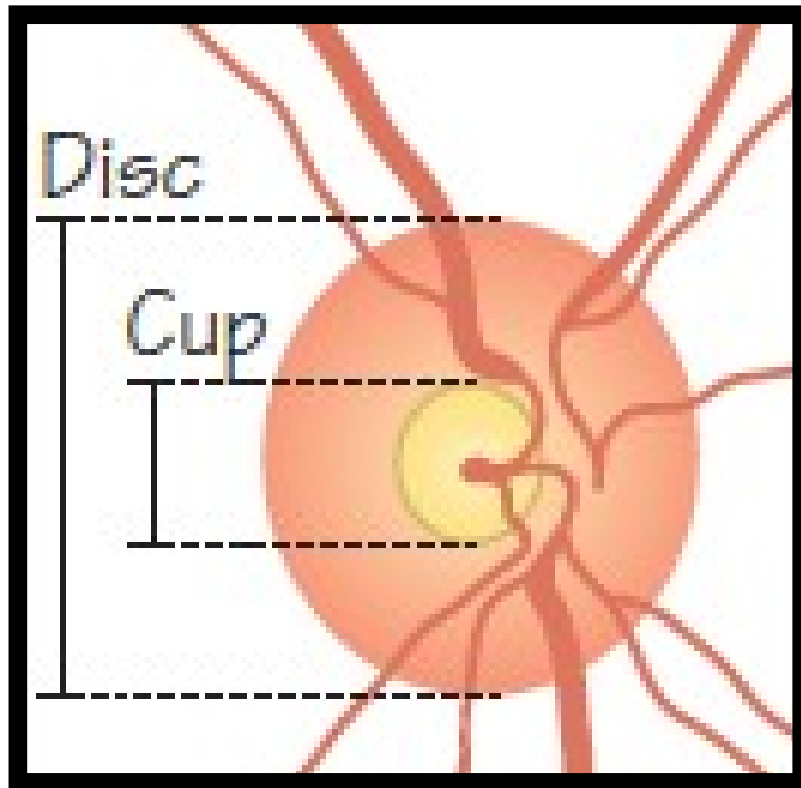


Goldman, Applanation
Tonometer
(On slit lamp)



Schiotz, Indentation
Tonometer
(On bed)

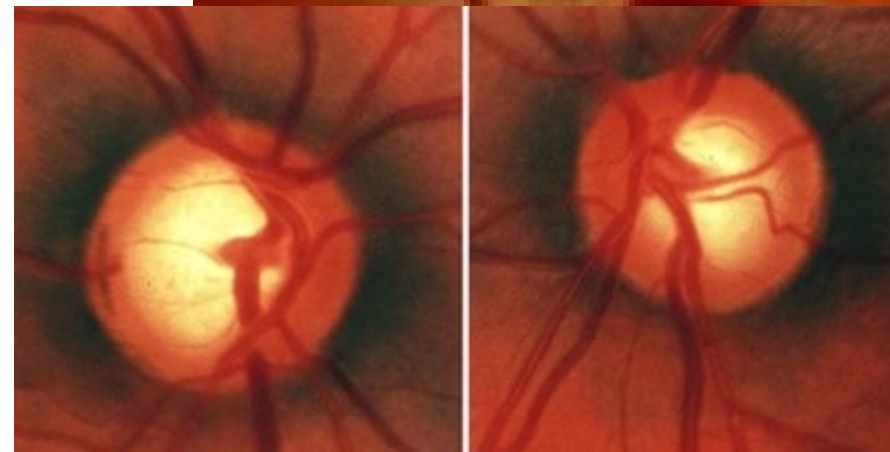
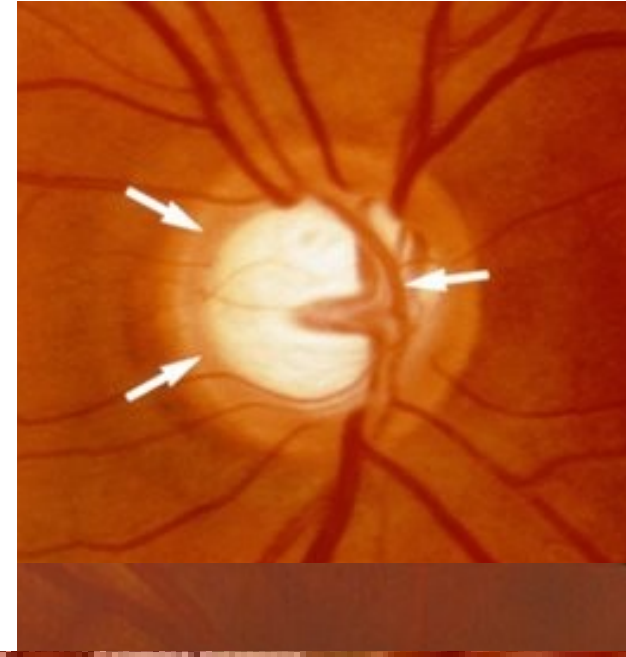
THE OPTIC NERVE HEAD:



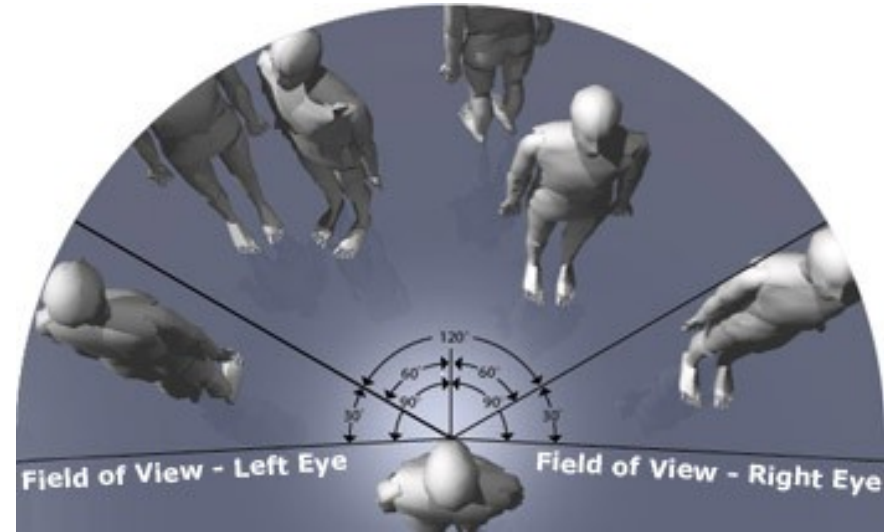
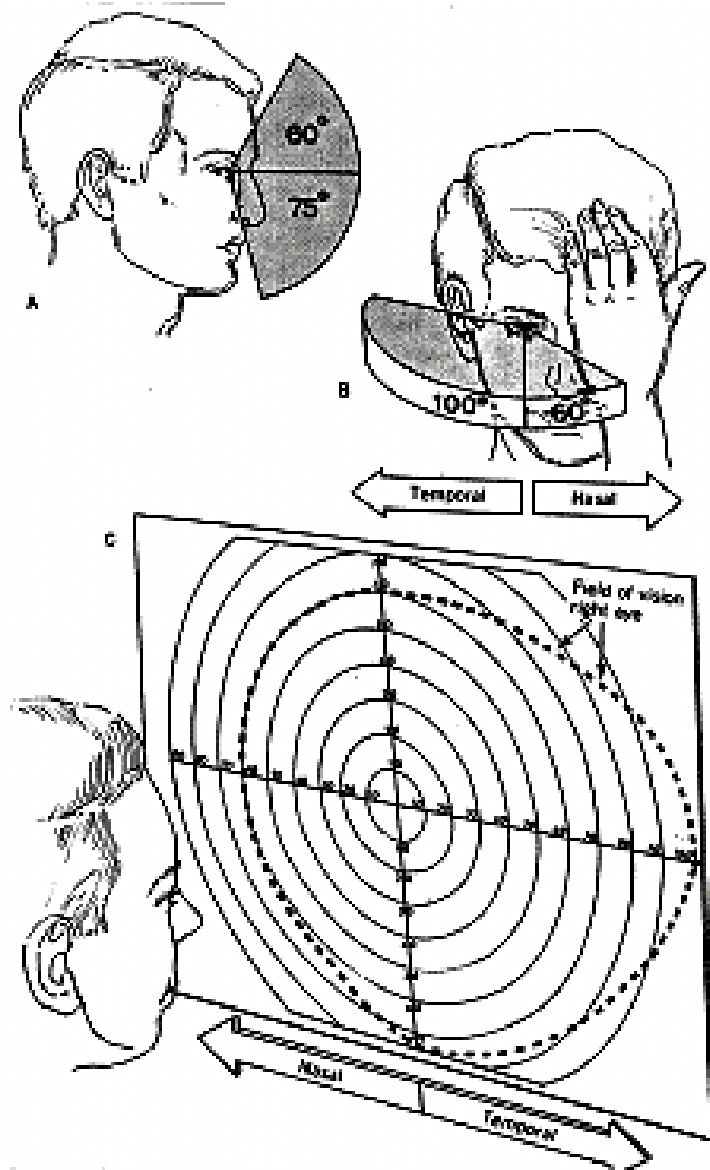
Normal Optic Nerve Head

- Disc changes in Glaucoma:

- Increased cup / disc ratio.
- Vertical elongation of cup
- Visibility of pores of lamina cribrosa
- Nasal deflection of disc vessels
- Bayonetting , notching
- Splinter hemorrhage
- Peripapillary pigmentation
- Cup asymmetry



FIELD OF VISION

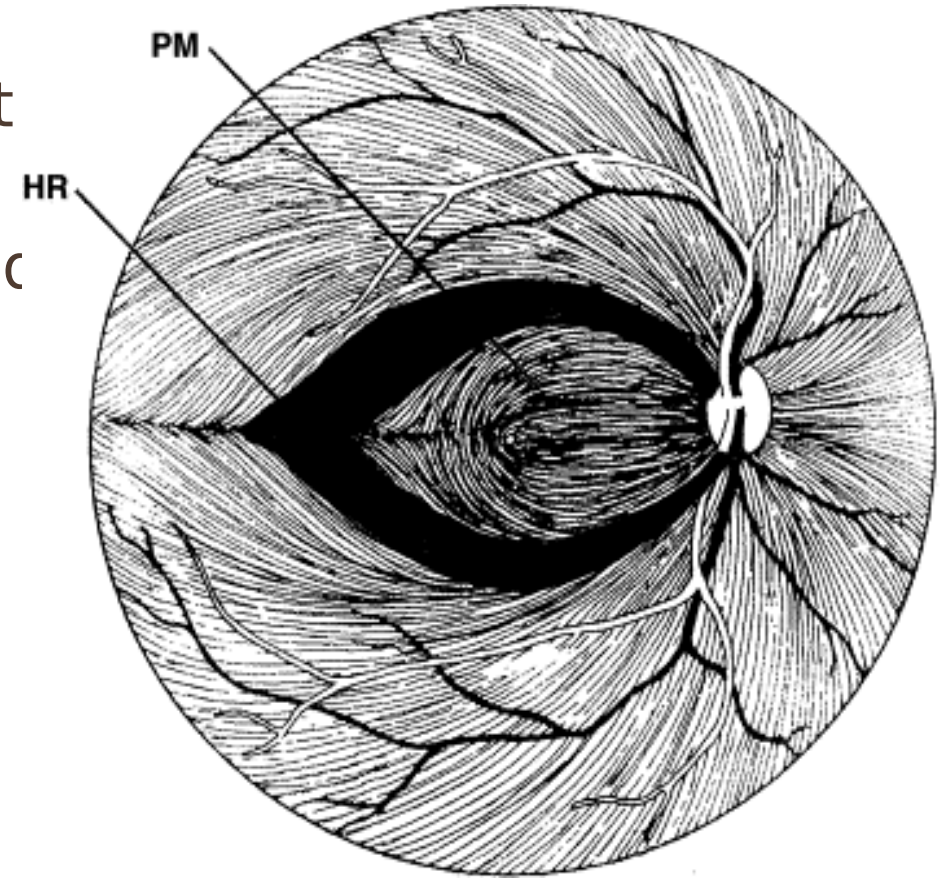


HOW TO TEST FOR VISUAL FIELD? “PERIMETRY”

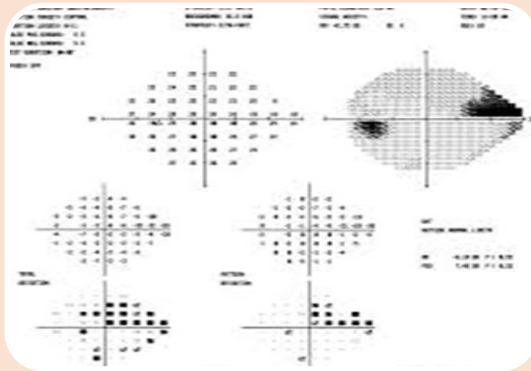


VISUAL FIELD DEFECTS:

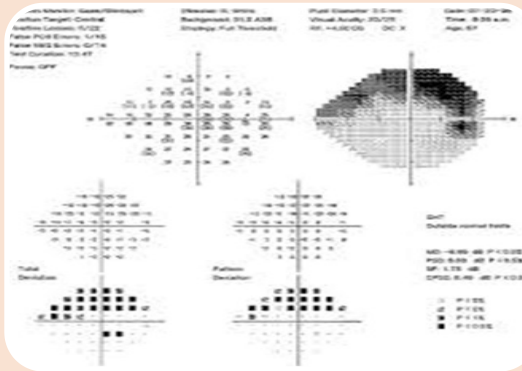
- ▮ The earliest fibers to be affected is the most crowded fibers?
- ▮ The latest to be affected is the least crowded fibers?
- ▮ Progression; very **important**
 - ▮ **Scattered** central scotoma in **arcuate areas**
 - ▮ **Complete** arcuate scotoma
 - ▮ **Nasal step**
 - ▮ **Macula late**



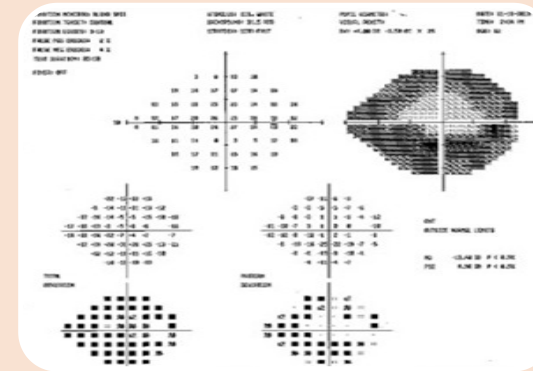
VISUAL FIELD CHANGES IN GLAUCOMA:



The earliest clinically significant defect is an isolated **scotoma**



The scotoma elongates circumferentially along the distribution of the arcuate nerve fibers forming an upper or lower **arcuate**



Upper and lower - arcuate scotomas are present, they form a **double arcuate**

Contraction of - the peripheral field that usually spares the central vision until late in the disease

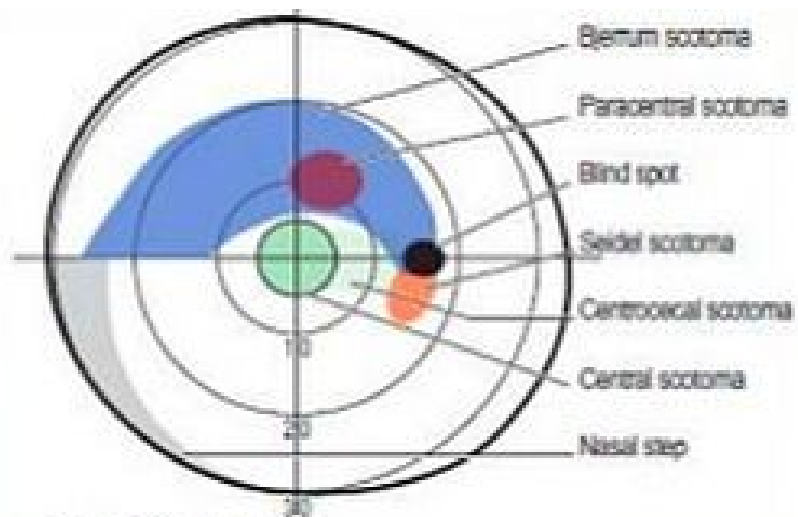
TYPES OF VISUAL FIELD DEFECTS IN GLAUCOMA:

- Visual field defects leads to

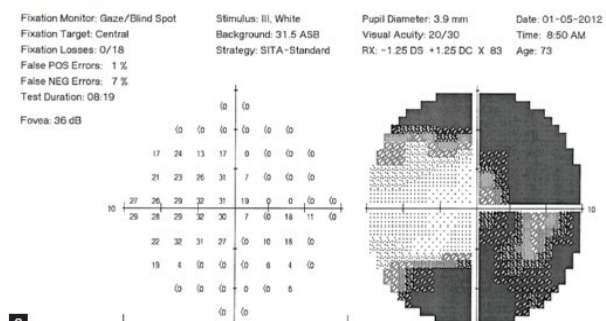
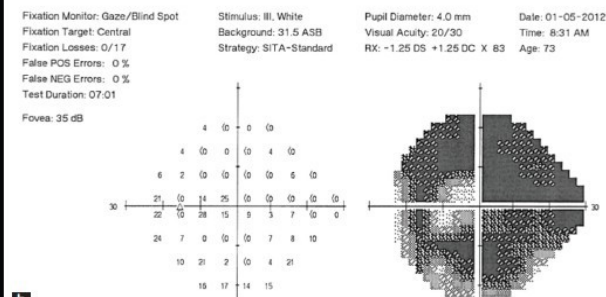
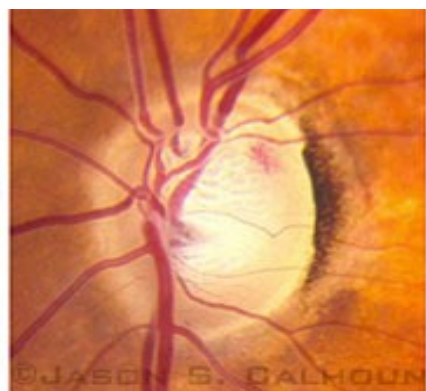
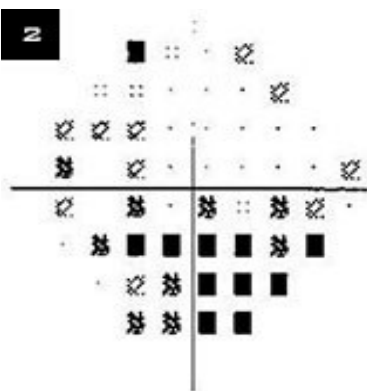
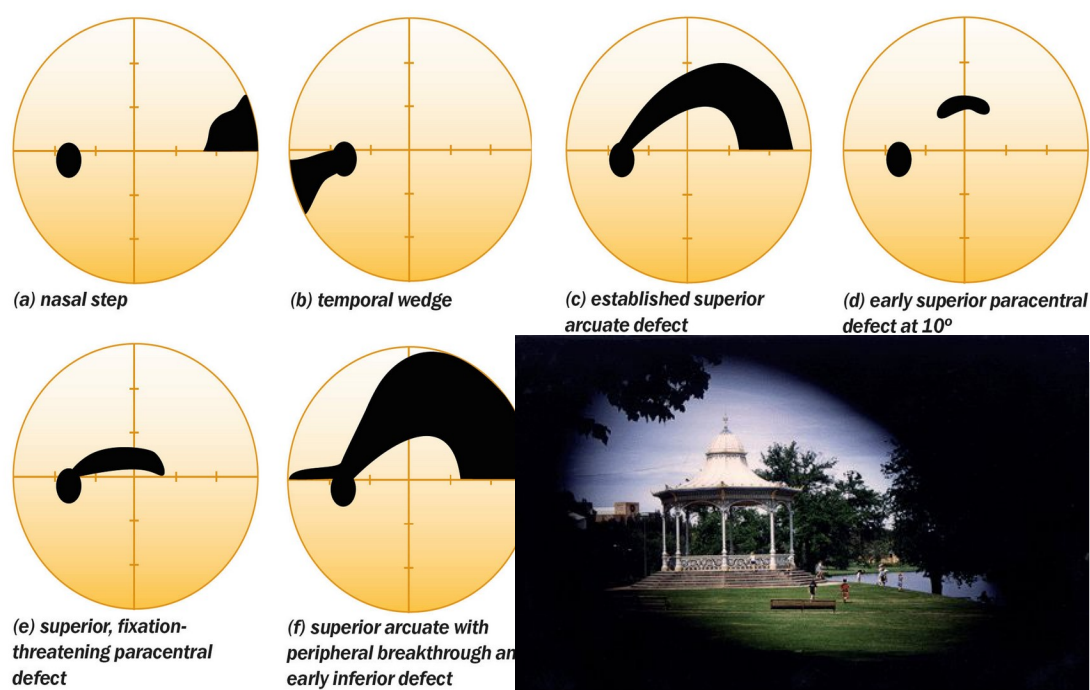
- SCOTOMA**

Area of **reduced visual acuity** surrounded by field of normal vision

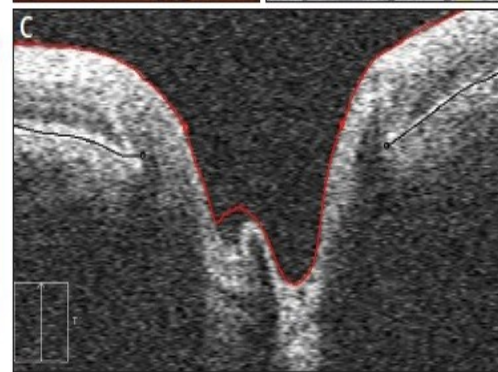
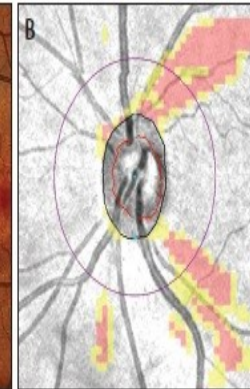
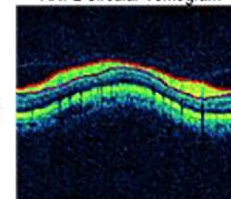
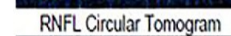
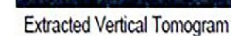
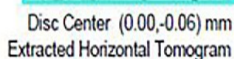
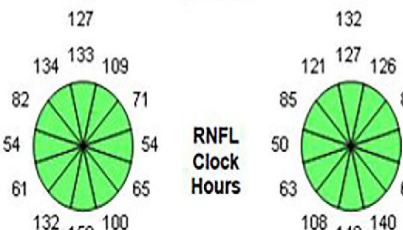
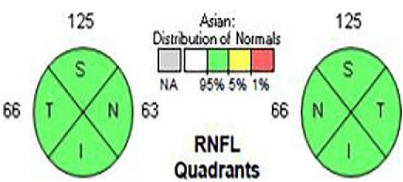
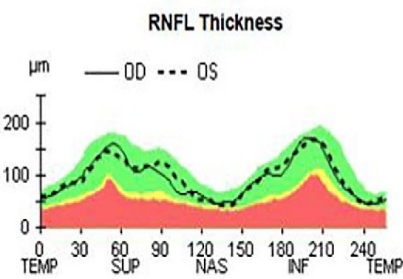
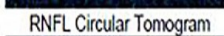
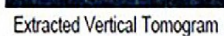
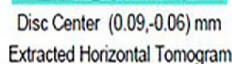
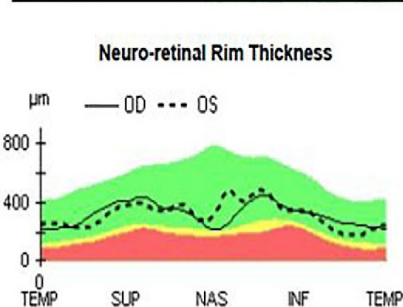
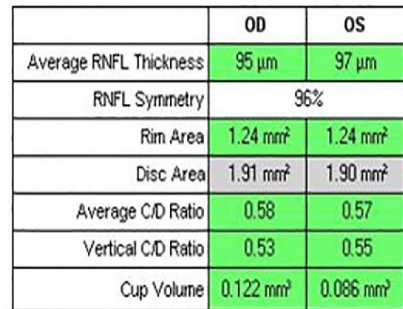
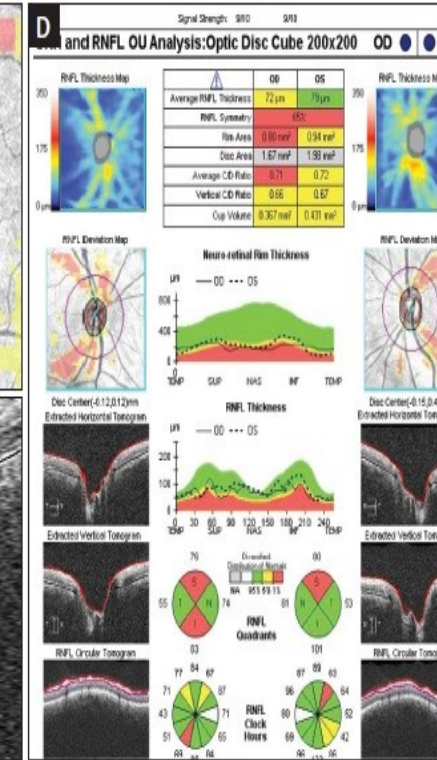
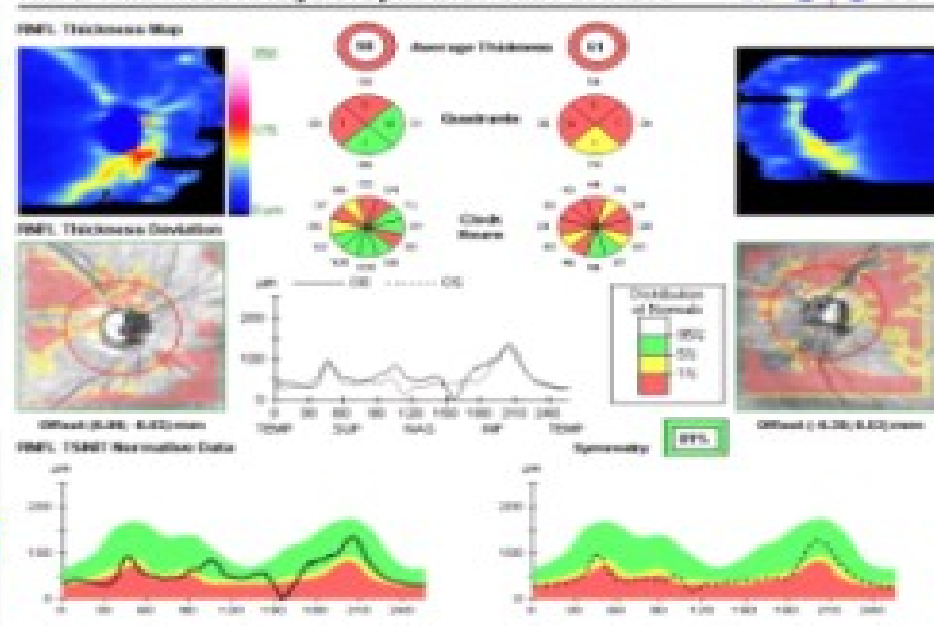




C Types of scotoma



OD	●	●	OS
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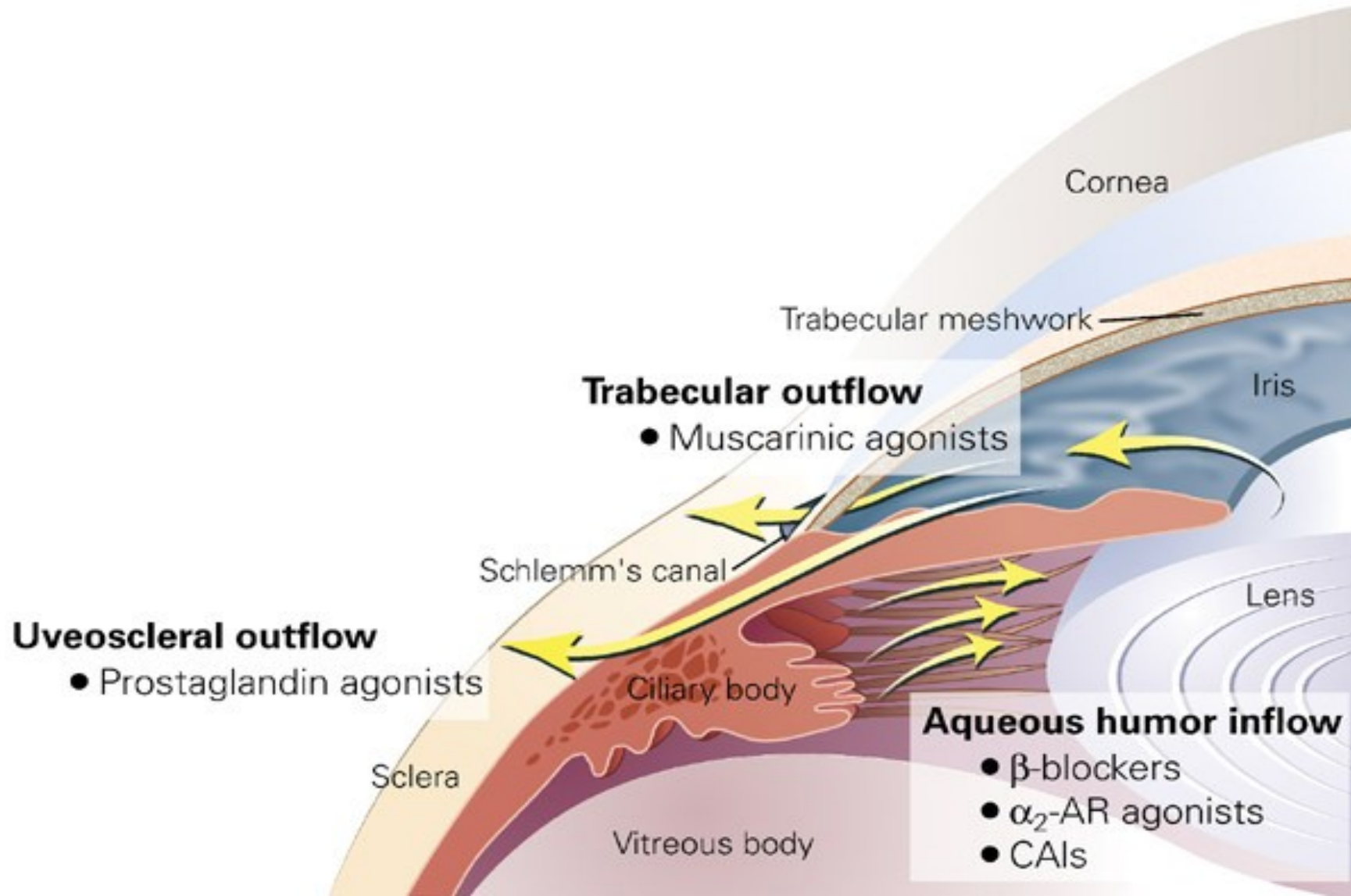
**RNFL Thickness Analysis: Optic Disc Cube 200x200**

TREATMENT OF POAG:

Medical



MECHANISM OF ACTION OF ANTI-GLAUCOMA DRUGS



- Medical treatment
 - Start medical
 - Start Prostaglandin analogue
 - Pressure reduction by at least **30%** (target pressure)
 - Follow up
 - Tension every 3 months
 - Field every 6 months
 - OCT yearly
 - When to stop and change ?
 - Non-adherence , non compliant
 - Negligence
 - Socio-economic
 - Progression

TYPES OF ANTIGLAUCOMA MEDICATIONS:

▮ First choice antiglaucoma medication:




Prostaglandin analogues

Latanoprost (Xalatan)

Mode of action	Dose	Local side effects	Systemic side effects
• Increase uveo-scleral out flow	1x1	<ul style="list-style-type: none">• Iris pigmentation• Increase lid pigmentation• Overgrowth of lashes• Iritis• Cystoid macular edema	<ul style="list-style-type: none">• No systemic side effects


TYPES OF ANTIGLAUCOMA MEDICATIONS:

▮ First choice antiglaucoma medication:

	Mode of action	Dose	Local side effects	Systemic side effects
<u>Beta blockers</u> (Timolol Maleate)	<ul style="list-style-type: none">• Decrease aqueous formation by 40%• Act on beta 1 and beta 2 receptors	1x2	<ul style="list-style-type: none">• Allergy• Mild dry eye	<ul style="list-style-type: none">• Worsening bronchial asthma• Hypotension• Masking sympathetic response to hypoglycemia• Worsening of lipid profile• Depression• Insomnia• Impotence


TYPES OF ANTIGLAUCOMA MEDICATIONS:

▮ Second choice antiglaucoma medication:

 <p>Alpha-agonist</p> <p>(Brimonidine)</p> <p>Alphagan-P</p>	Mode of action	Dose	Local side effects
	• Decrease aqueous formation by acting on the inhibitory alpha receptors	1x2	• Allergy • Hyperemia

TYPES OF ANTIGLAUCOMA MEDICATIONS:

▮ Second choice antiglaucoma medication:


	Mode of action	Dose	Local side effects
Miotics (Pilocarpine nitrate 1-4 %)	They stimulate muscarinic receptors in the ciliary muscle pulling on the trabecular meshwork increasing the aqueous outflow.	1x2	<ul style="list-style-type: none">• Miosis• Ciliary spasm• Headache• Iritis

TYPES OF ANTIGLAUCOMA MEDICATIONS:

▮ Second choice antiglaucoma medication:

Topical carbonic anhydrase inhibitor: (Dorzolamide)

They inhibit the aqueous formation in the ciliary body.

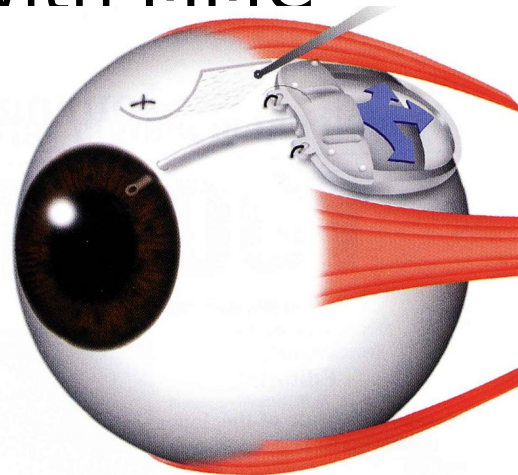
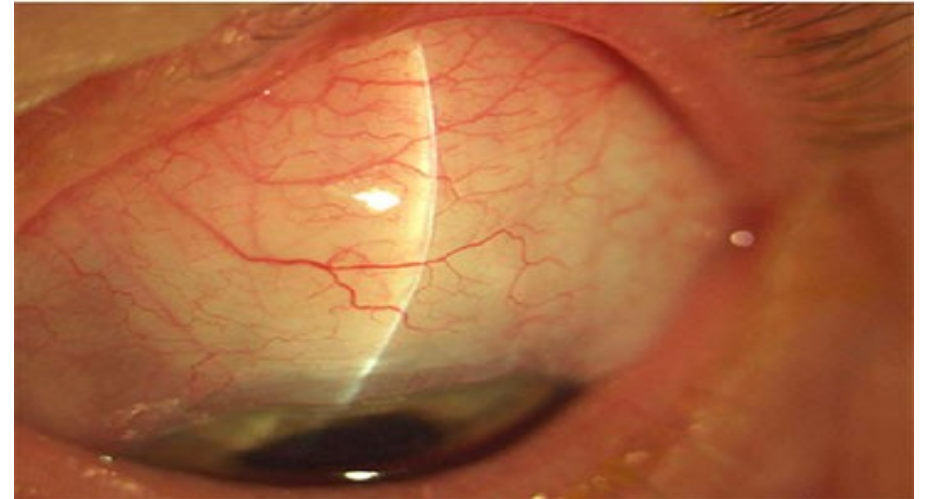
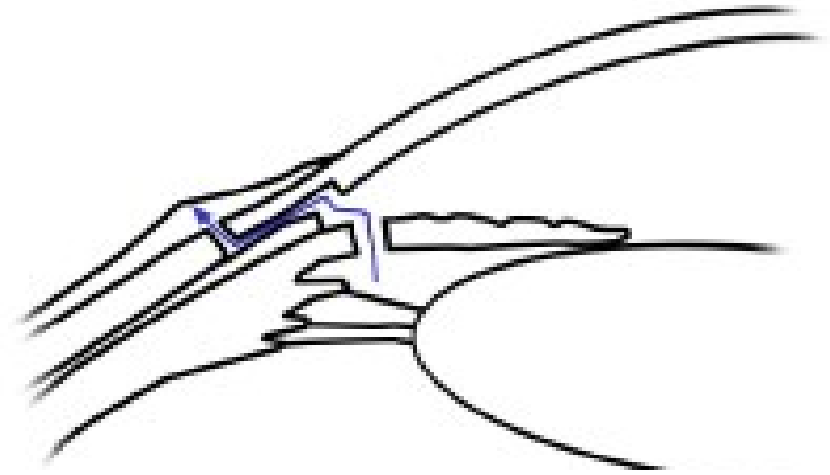
Drug	Mode of action	Dose	Systemic side effects
 Systemic carbonic anhydrase inhibitor. (Acetazolamide) Cidamex	<ul style="list-style-type: none">• Decrease aqueous formation	250 mg/6 hrs. for short term	<ul style="list-style-type: none">• Tingling & numbness• GIT upset• Renal stones

SURGICAL TREATMENT:

▮ Indication:

- 1- Progressive nerve fiber layer damage, progressive cupping and/or progressive visual field loss inspite of the maximally tolerated medications
- 2- Non compliance of the patient to medical ttt
- 3- Intolerance to the medical ttt

- Laser treatment
 - Selective laser **trabeculoplasty**
 - 15% reduction
 - Poor in younger age
 - Temporary (3-5 yrs.)
- Surgical treatment
 - **Trabeculectomy** with MMC
 - MIGS
 - Setons



SECONDARY OPEN ANGLE GLAUCOMA:

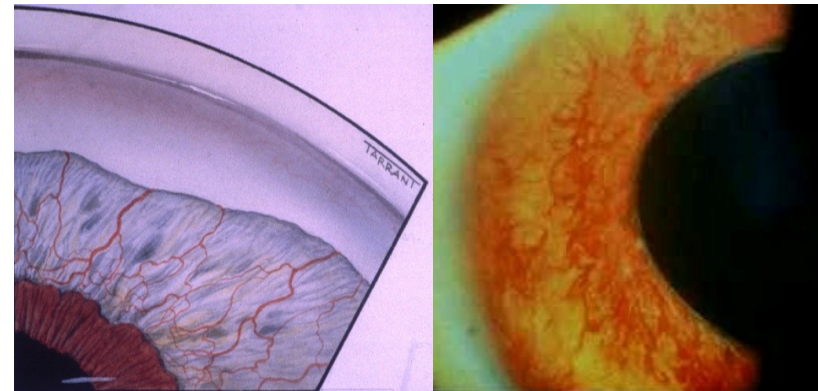
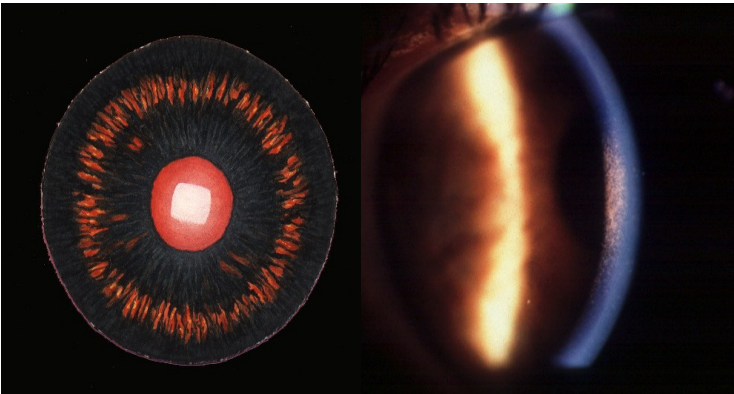
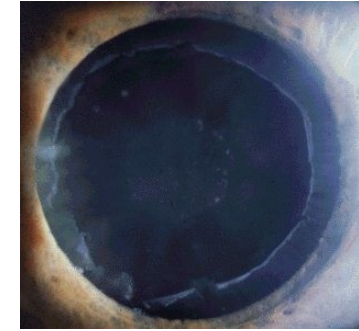
- ▮ IOP above 21 mmHg
- ▮ Optic disc cupping
- ▮ Glaucomatous field changes
- ▮ Open angle on gonioscopy



A Cause

Chronic Glaucoma (Secondary)

- Pseudoexfoliation glaucoma
- Pigmentary glaucoma
- Steroid-induced glaucoma
- Angle recession (traumatic) glaucoma
- Post-operative glaucoma, steroid induced
- NVG



LENS INDUCED 2RY OPEN ANGLE GLAUCOMA:

▮ Phacolytic glaucoma:

- In **Hypermature cataract**
- The TM is blocked by the lens proteins engulfed by the macrophages

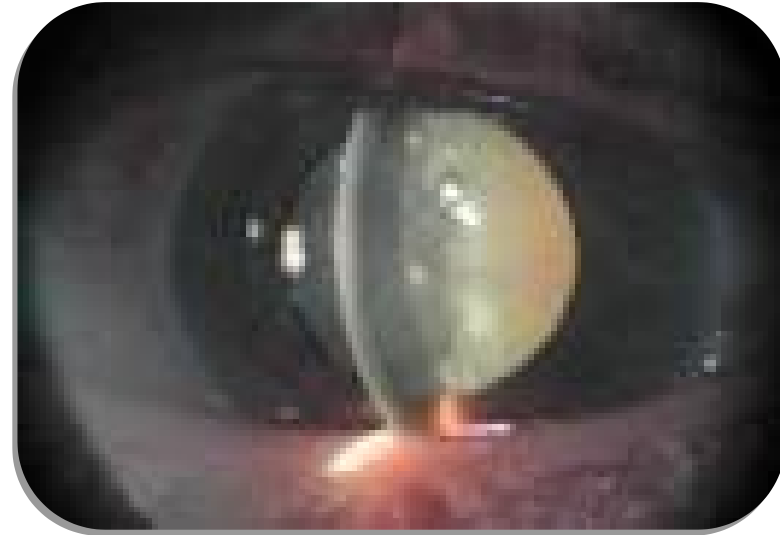


▮ Phacoanaphylactic glaucoma:

- An autoimmune reaction which occurs when a relatively large amount of lens matter is exposed to the aqueous after trauma or surgery

LENS-INDUCED 2RY ANGLE-CLOSURE GLAUCOMA:

- ▮ Phacomorphic glaucoma:
 - **In Intumescent cataract**
 - Leading to pupillary block and angle closure
 - Treated by **lens extraction**



TRAUMATIC GLAUCOMA:

▮ Hyphema:

- The TM is blocked by blood (RBCs)

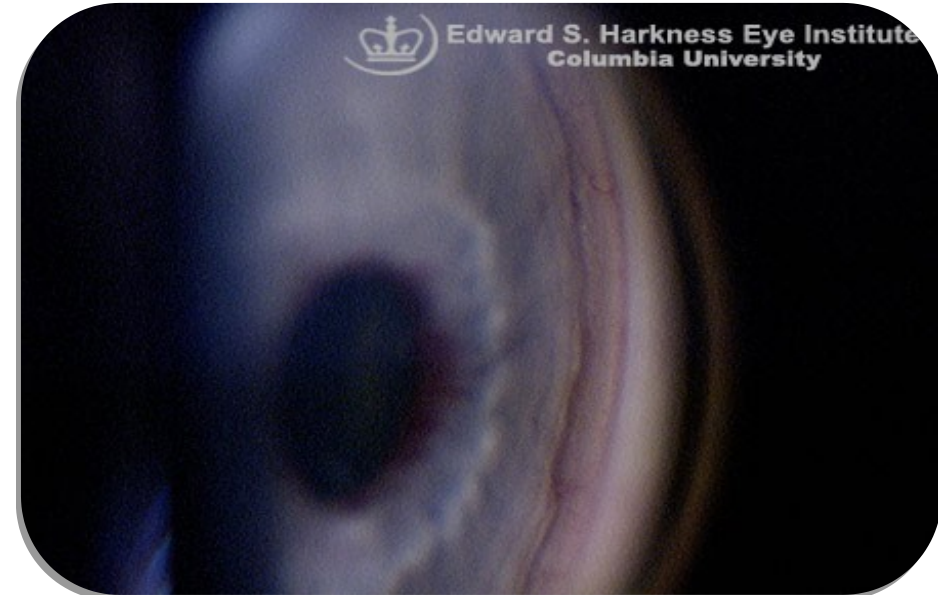


▮ Angle recession glaucoma:

- occurs several weeks to months after blunt trauma due to **fibrous changes** at the level of the draining channels for aqueous

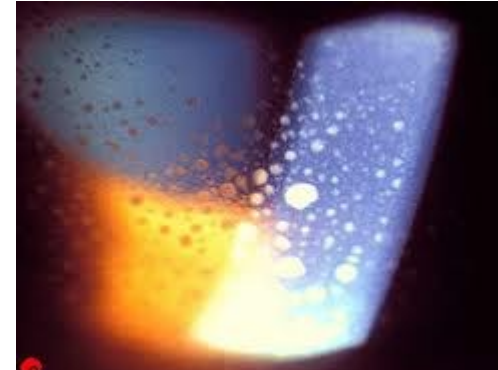
NEO VASCULAR GLAUCOMA:

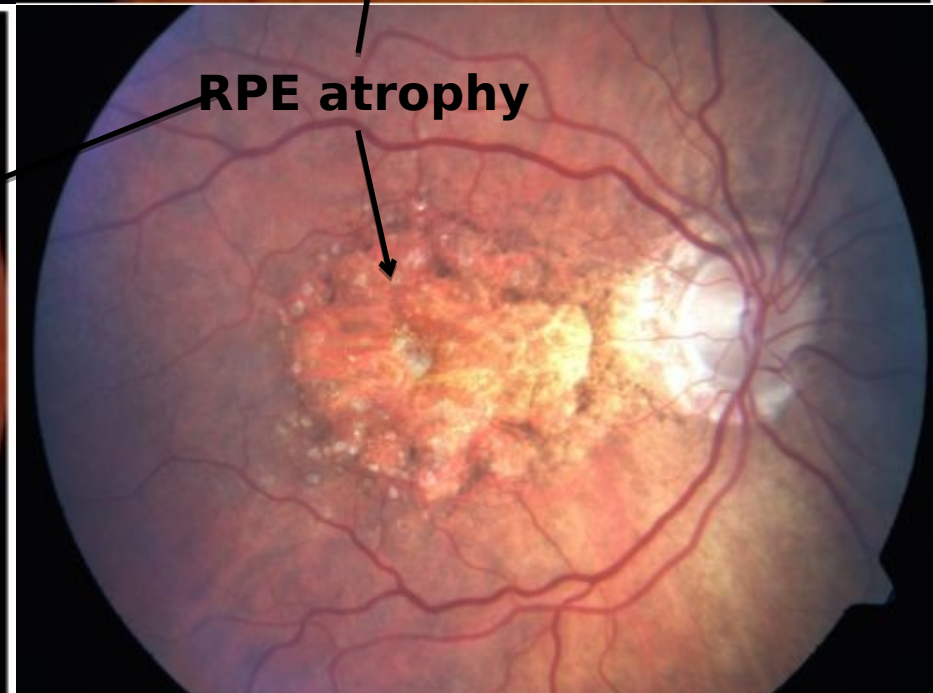
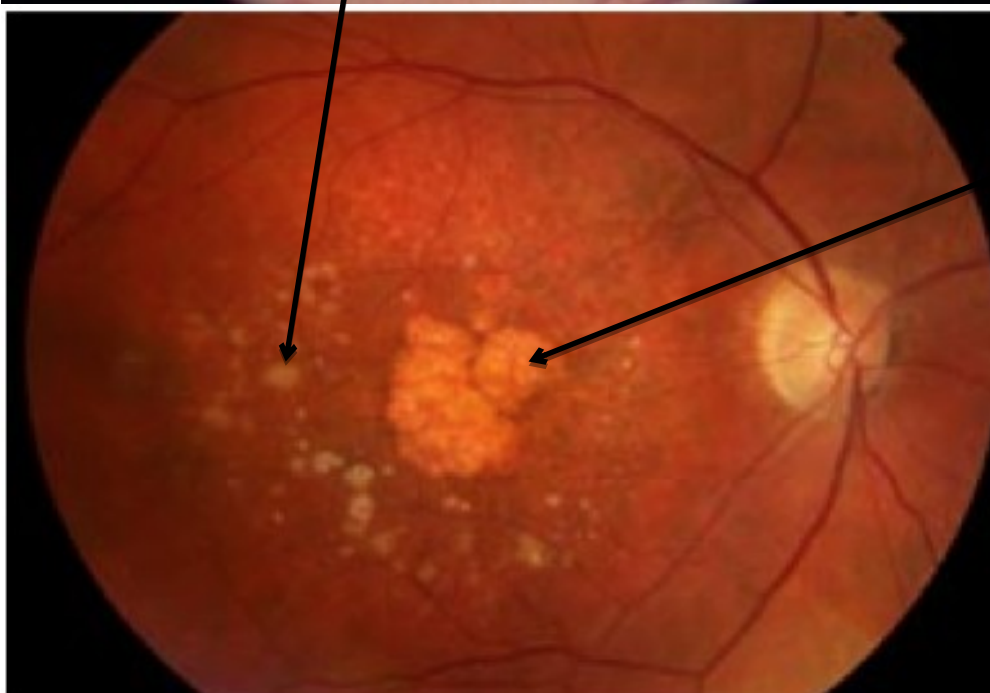
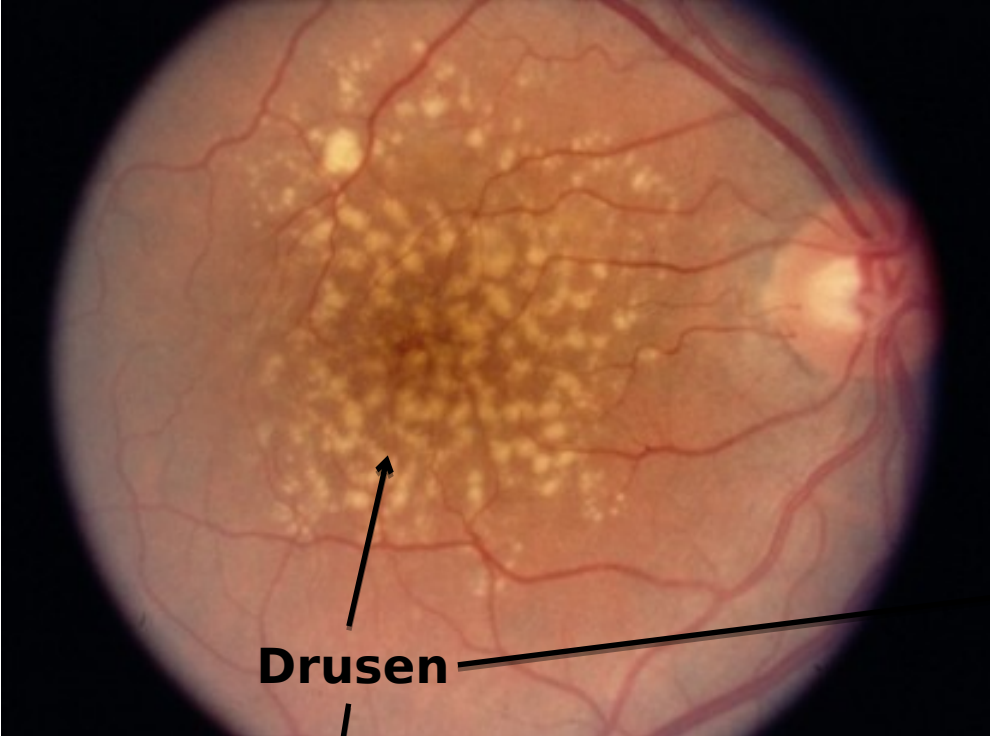
- ❑ **Ischemic retinal** conditions
- ❑ Abnormal vessels creep on the surface of the iris (**rubeosis irides**)
- ❑ Abnormal vessels encroach on the angle with leakage of proteins blocking the TM pores
- ❑ End stage : **synechial angle closure**
(PAS) peripheral anterior **synechial**



INFLAMMATORY GLAUCOMA

- **Uvietis:**
- TM is blocked by plasmoid aqueous.
- Trabeculitis (HZ) .
- Ring synechiae
(ocllusio pupillae)
pupillary block .



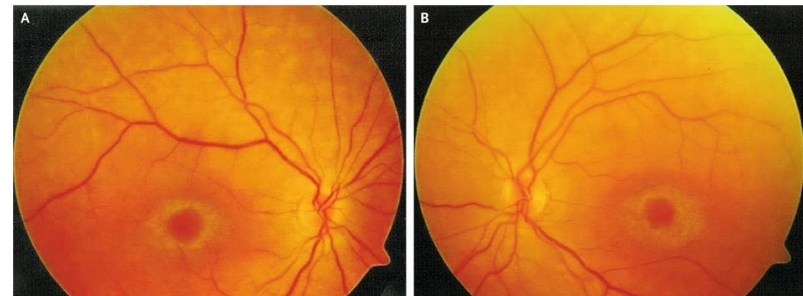


Retinal dystrophies

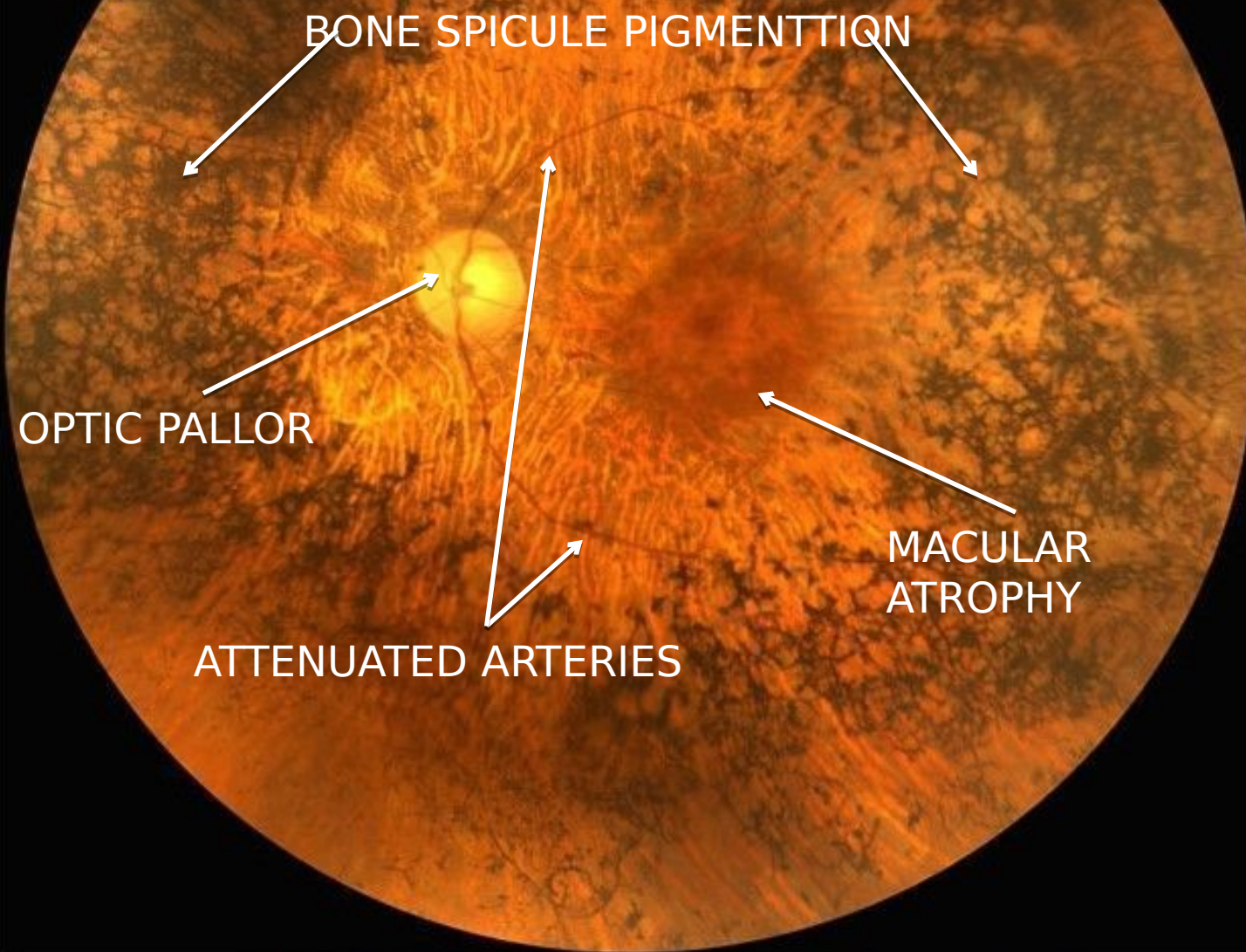
- Progressive degeneration of retinal photoreceptor cells of hereditary origin; AR, AD and X-linked
- Rod-cone dystrophies
 - Night blindness (**nyctalopia**), loss of peripheral field
- Cone-rod dystrophies
 - Decreased vision, **photophobia**, color weakness, central scotoma
- ERG: scotopic and photopic
- Treatment; **supportive, developing gene therapy**

Retinal dystrophies

- Primary Pigmentary Retinopathy
- **Rod-Cone dystrophy** : Night blindness (nyctalopia), loss or peripheral field
- **Cone-Rod dystrophy**: hereditary atrophy of the cones
 - Present with cone symptoms at young age
 - Loss of vision and color
 - Photophobia
 - Central scotoma
 - **Bull's eye maculopathy**
 - OCT, Photopic ERG and mfERG
 - **Only supportive treatment**



TYPICAL RETINITIS PIGMENTOSA



Pigmentary Retinopathy (Retinitis Pigmentosa)

- Hereditary defects in Rhodopsin and other related proteins resulting in early rod atrophy → night blindness at early age
- Reactionary RPE proliferation → **spider** (**bone corpuscle**) **pigment**
- Later progressive loss of vision (cone atrophy)
- **Annular scotoma**
- **Attenuated arteries** and pale disc (consecutive atrophy)
- Flat scotopic ERG

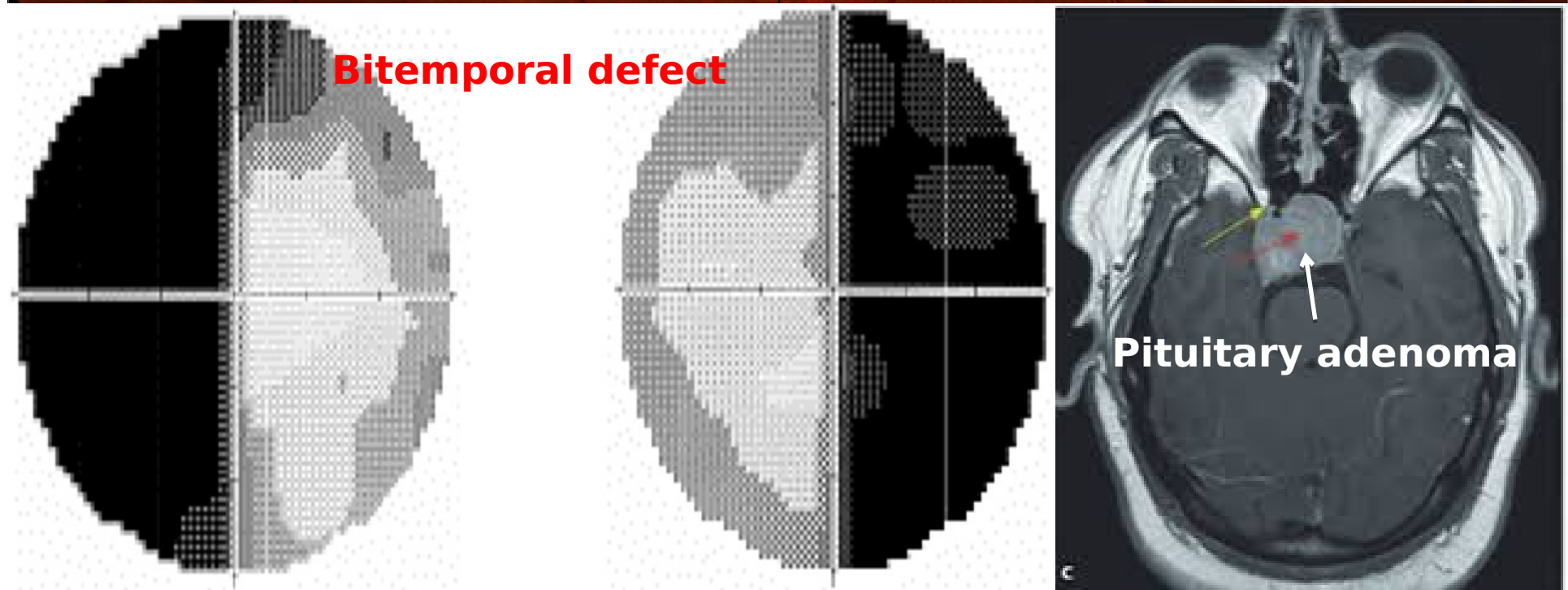
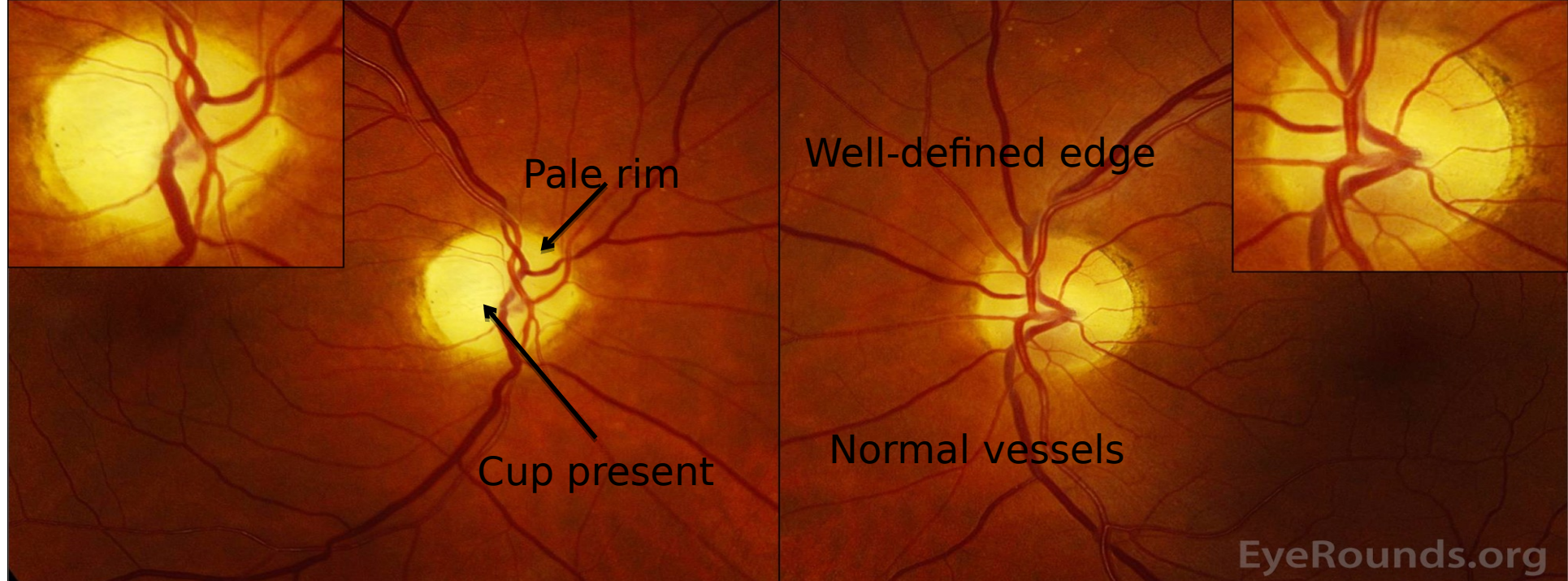
Syndromes with RP

- Usher
 - Deafness
- **Bardet-Biedl**
 - Truncal obesity
 - Hypogonadism
 - Polydactyly
 - Renal problems
- Refsum
 - Deficiency of phytanic oxidase
 - Cataract
 - Anosmia
 - Deafness
 - Peripheral neuropathy

- Abetalipoproteinemia
 - Defective fat absorption
 - Diarrhea
 - Ataxia
 - Acanthocytosis
 - Low serum cholesterol, absent apo-LDL
- Mitochondrial
 - MT-ATP6 gene
 - Neuropathy
 - Ataxia

Compressive optic neuropathy

- Progressive atrophy of the optic nerve as a result of retrobulbar compressive lesion
- **Orbital compression** and **TED**
- Pituitary and sellar lesion
- Clinical picture: optic nerve symptoms/signs
- **Field changes**
- **MRI**



Chronic papilledema

- Progressive loss of vision and field in chronic papilledema
- Pace depends on level of csf pressure
- IIH
- Secondary
- Field changes
- Management

Early papilledema

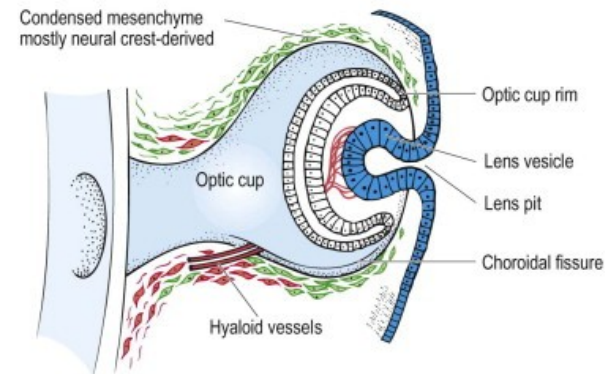
Established papilledema

Chronic papilledema

Atrophic papilledema

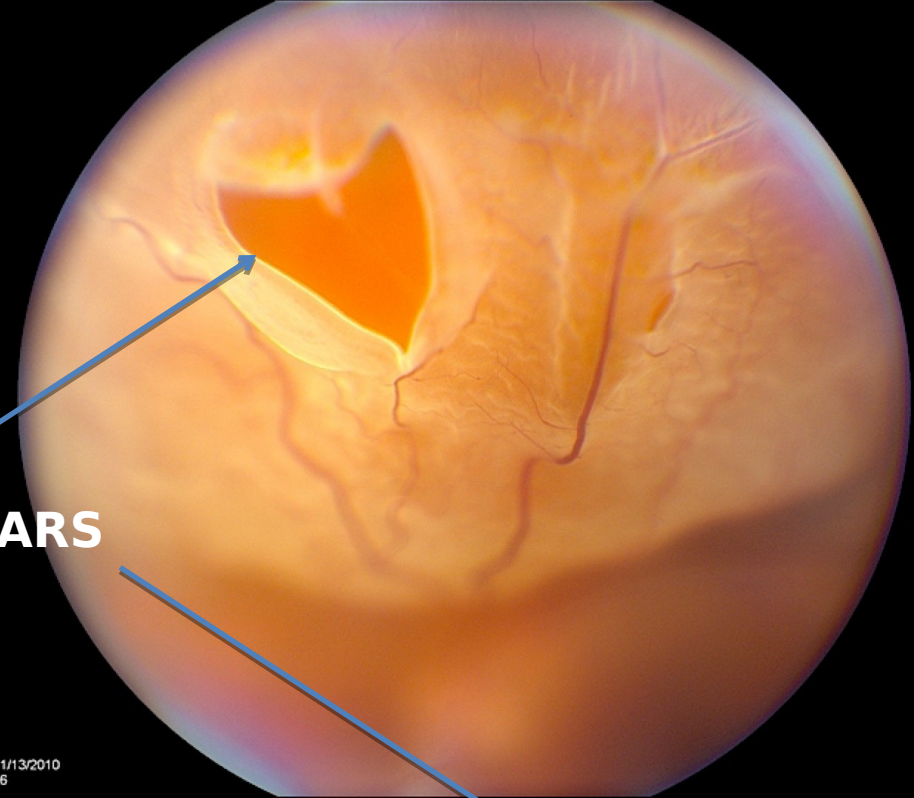
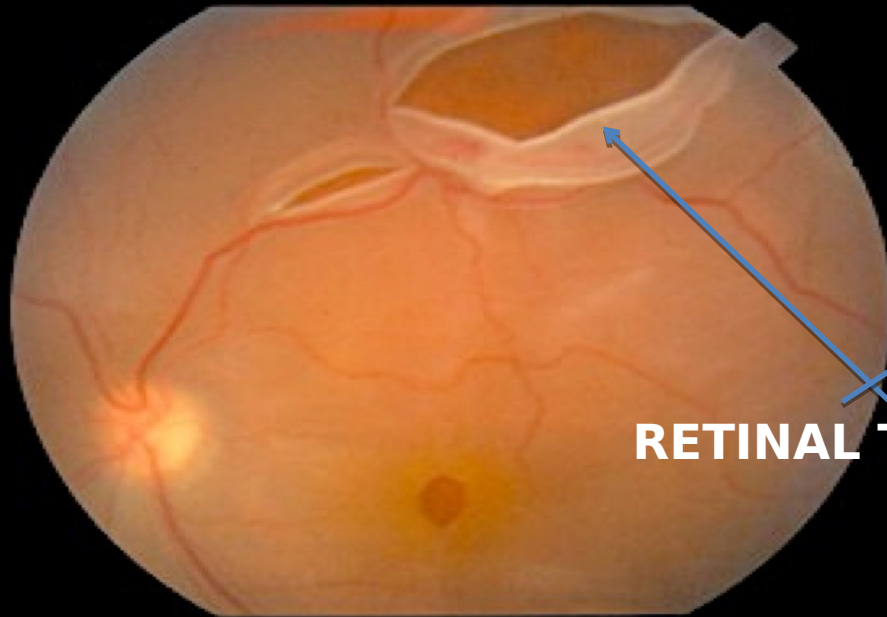
Retinal detachment (RD)

- Retinal detachment is the separation of the retina from the choroid
- Embryologically the retinal pigment epithelium (RPE) is the outer layer of the optic cup and firmly adherent to the choroid
- The inner retinal layers (sensory retina) are the inner layer of the optic cup and there is a potential space (between inner and outer layer of cup)
- So what happens in RD is the separation of the sensory retina from the RPE (sensory retinal detachment)

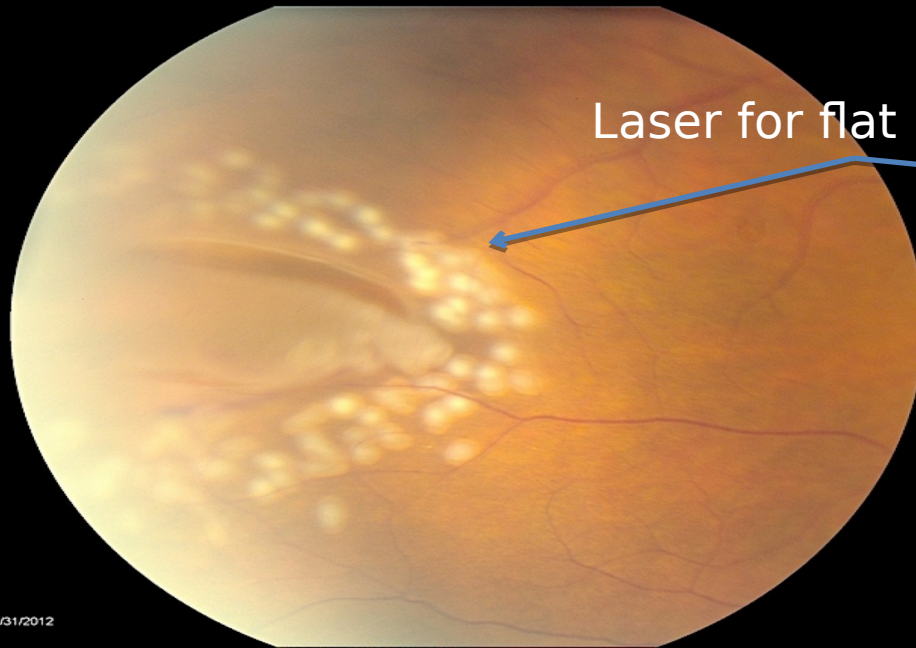


Retinal detachment : Mechanisms

- RD can result from several limited mechanisms
 - **Retinal tear** (tear=rhegma in latin): allows fluid vitreous to enter the potential space and separate the neurosensory retina. This is called **PRIMARY** or **RHEGMATOGENOUS** retinal detachment.
 - **Exudation** from the vascular choroid across the RPE: this is called **EXUDATIVE** retinal detachment and is seen in hypotony (after trauma or glaucoma surgery), choroidal melanoma and Haraada uveitis. When limited to the macular area it is called **SENSORY** RD
 - **Traction** from the vitreous: formation of fibrous strands in the vitreous that pull the retina causing **TRACTIONAL** RD; this is seen in proliferative DR and perforating trauma

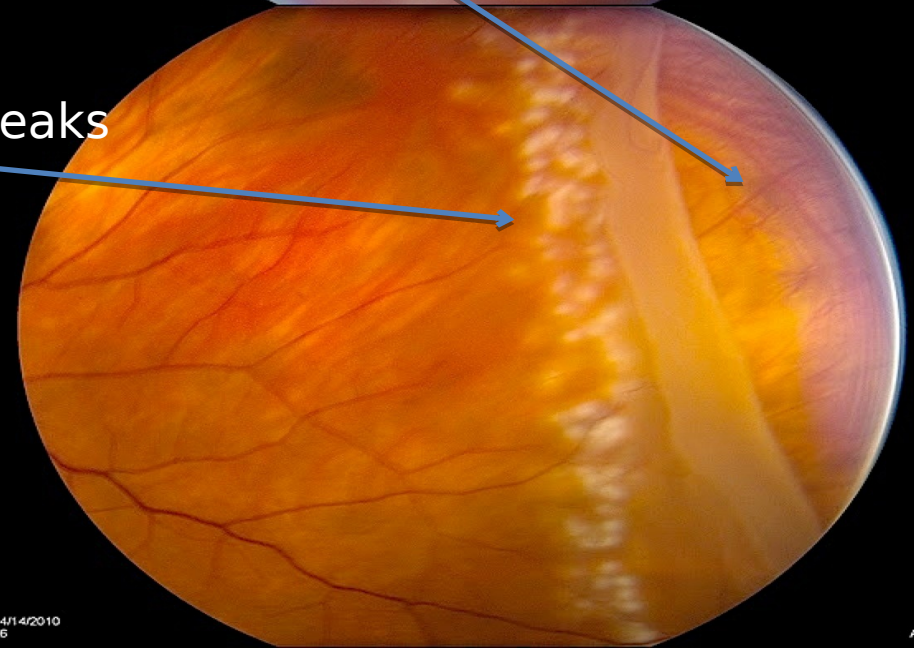


RETINAL TEARS



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E



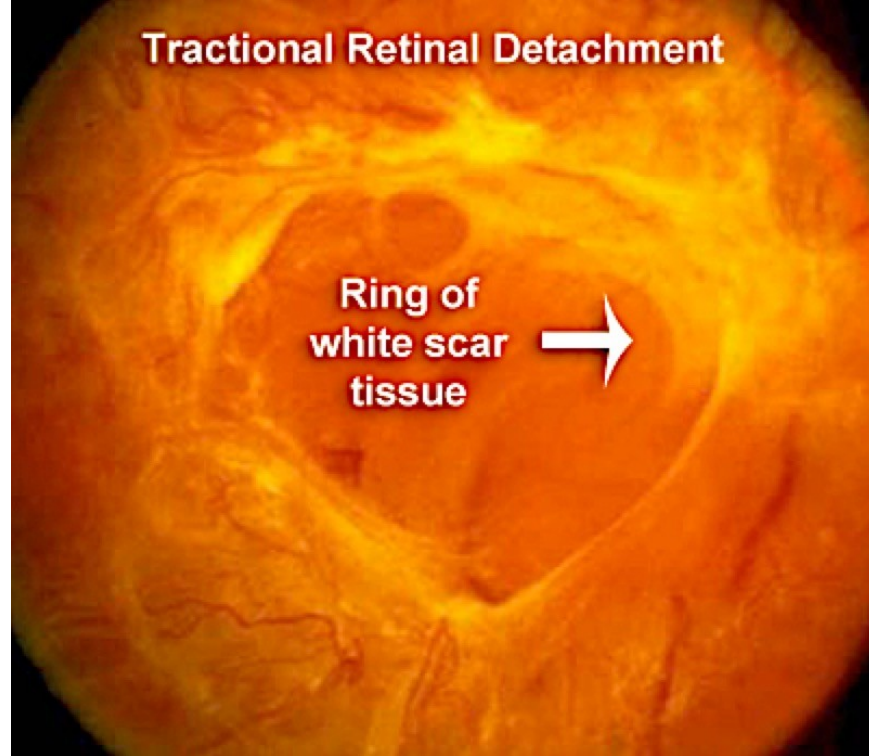
Laser for flat breaks

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1

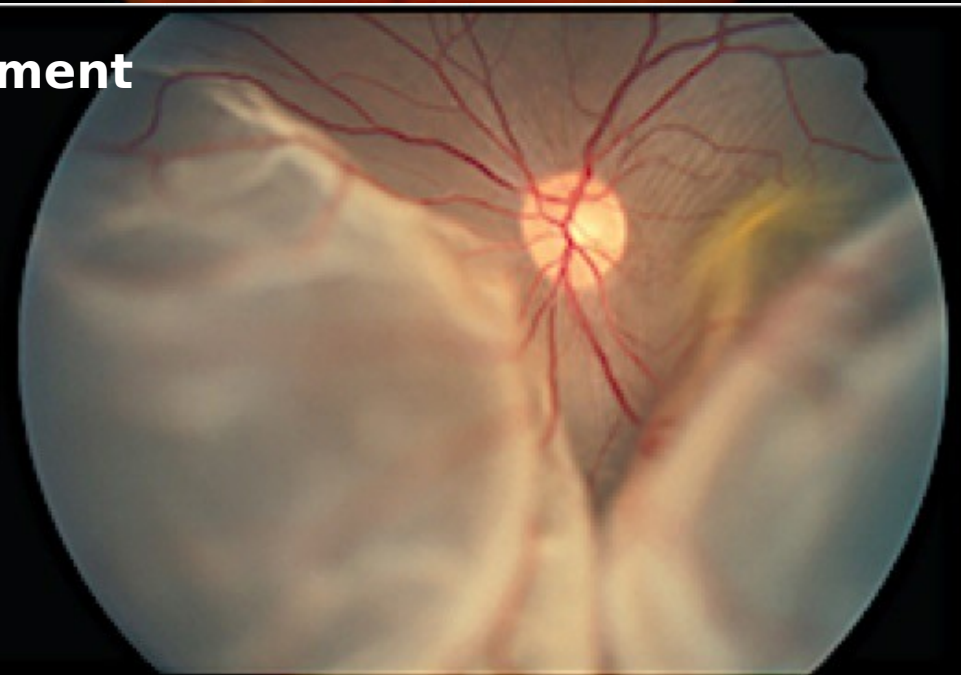
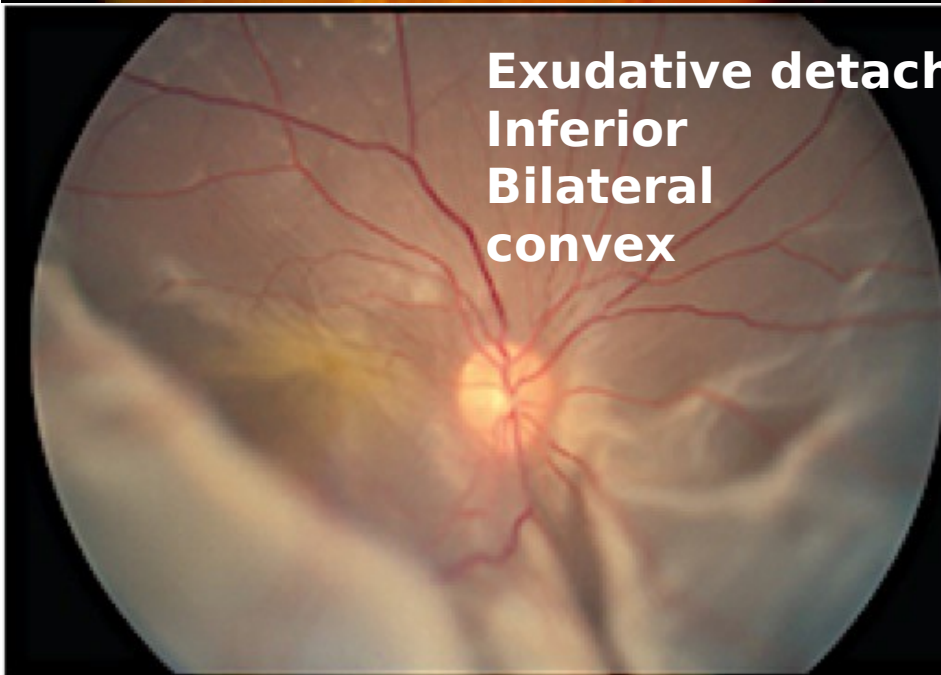
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AE

Tractional Retinal Detachment



**Exudative detachment
Inferior
Bilateral
convex**



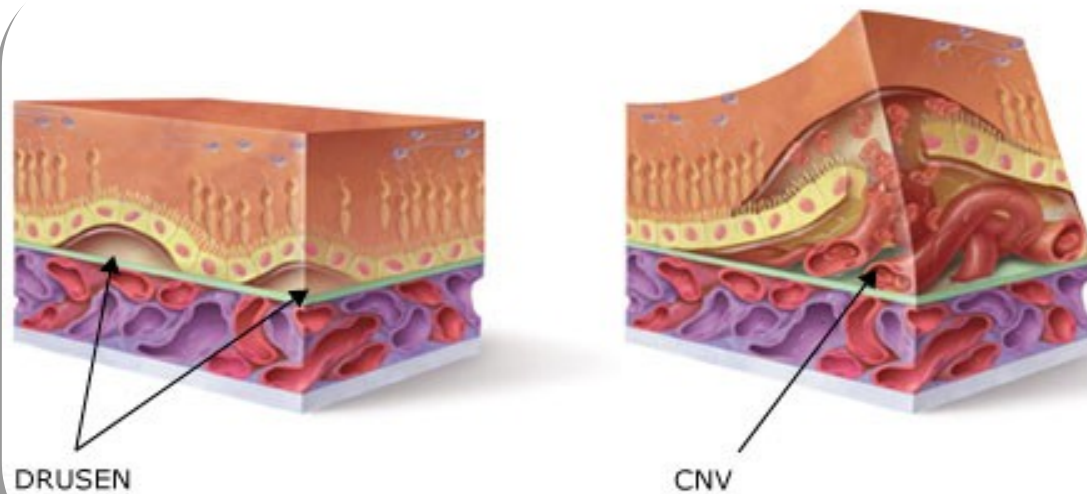
AMD Presentation aged macular DEGENERATION

Dry

Non-exudative ,
Atrophic ,
Non-neovascular.

Wet

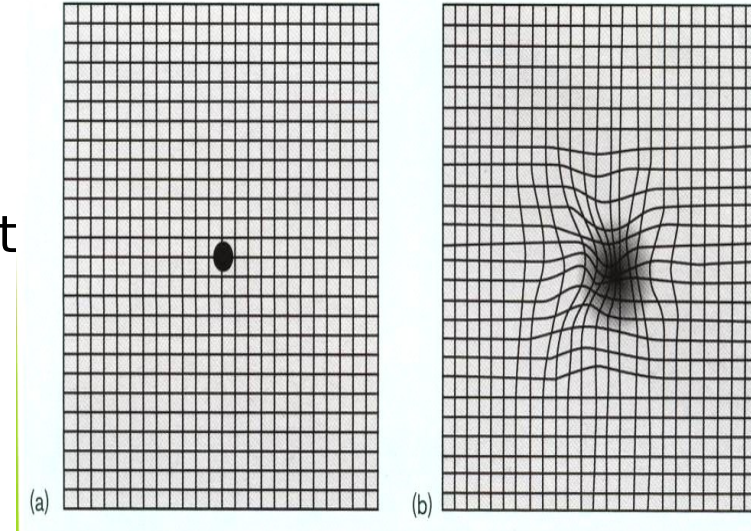
Exudative , Neovascular.



Dry AMD

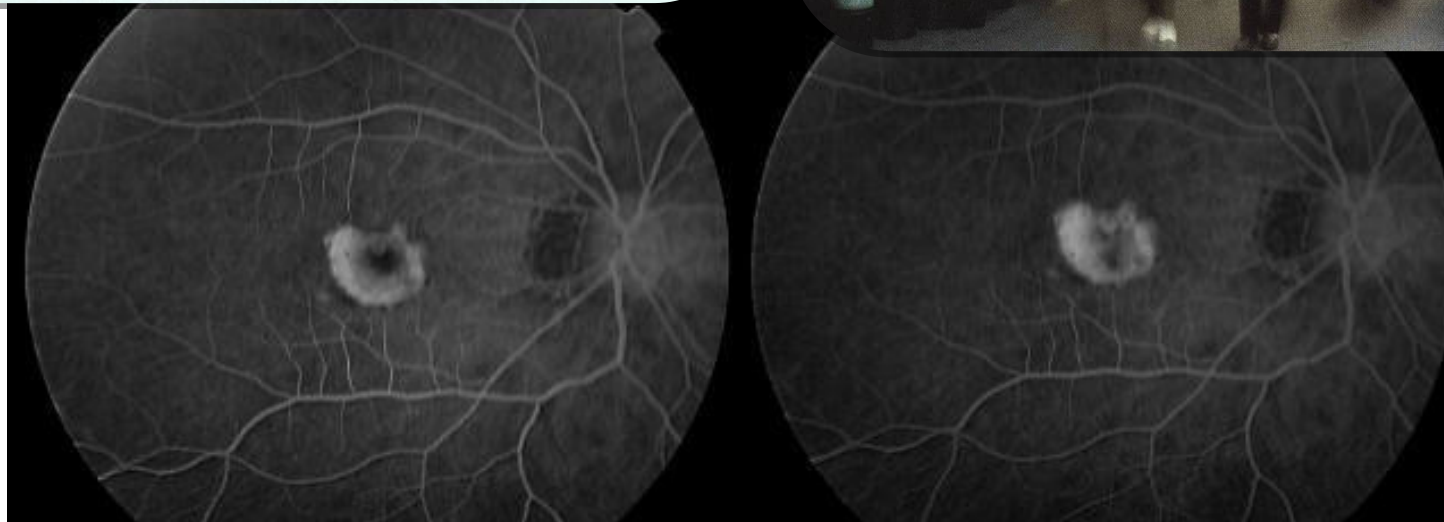
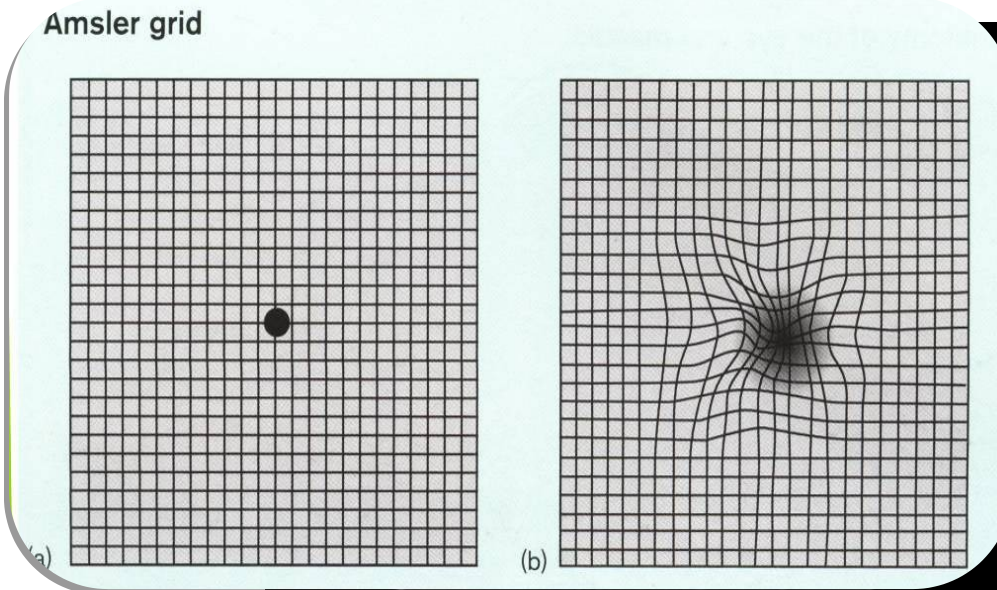
- Progressive atrophy of the photoreceptors and RPE in the macular area that worsens with age
- Phototoxicity and genetic predisposition
- May be of 2 types
 - Dry
 - Wet; acute onset of CNV
- Symptoms; progressive loss of vision, central scotoma
- Signs;
 - numerous Drusens
 - geographic macular degeneration
- FFA
- OCT
- **Wet AMD** ; CNV complication → **metamorphopsia**, hemorrhage and exudation
- Treatment; **supportive**

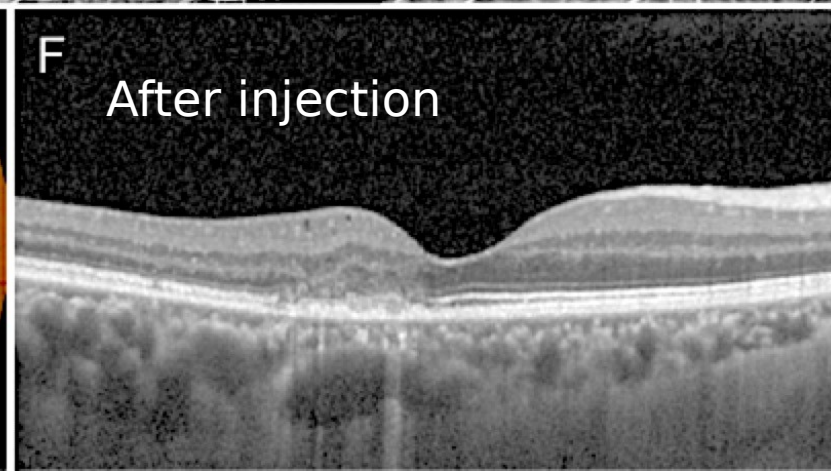
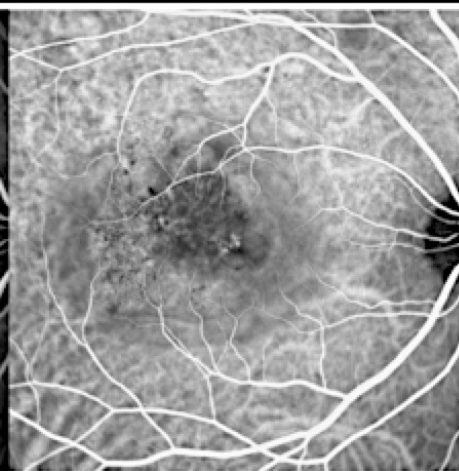
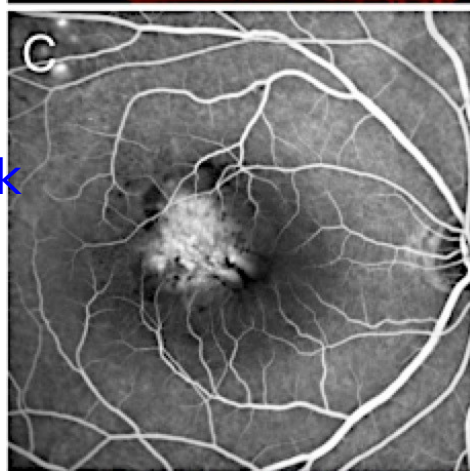
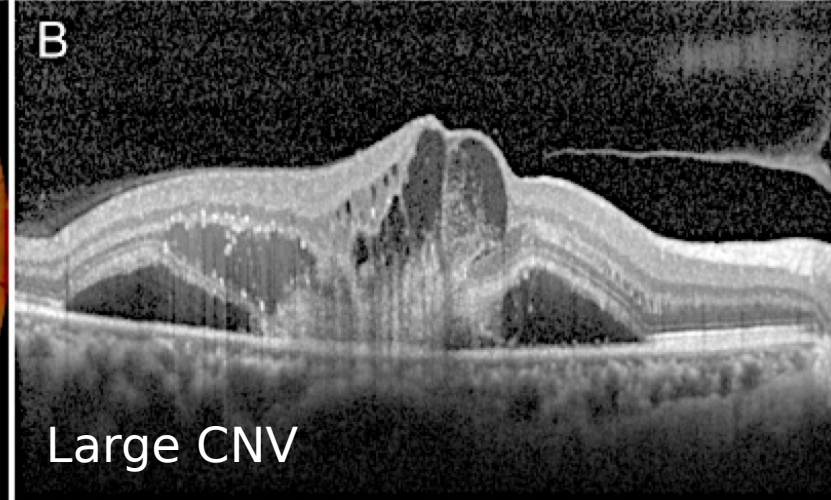
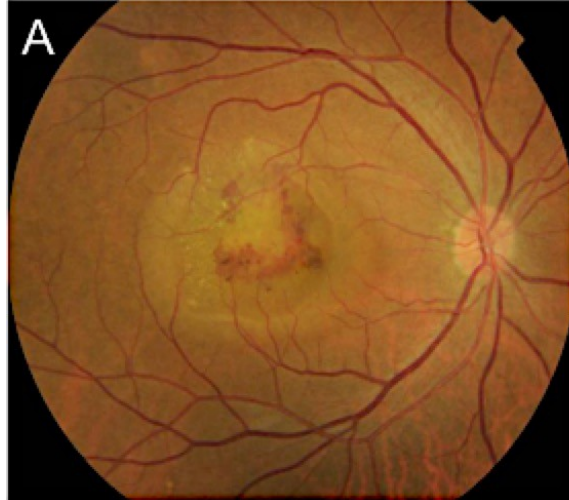
Amsler grid



Wet AMD

Amsler grid





Fluorescein leak